Dissertation on

“A HOSPITAL BASED STUDY ON CLINICAL PROFILE AND OUTCOME OF CEREBRAL VENOUS SINUS THROMBOSIS”

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CERTIFICATE

This is to certify that the dissertation entitled. **"A HOSPITAL BASED STUDY ON CLINICAL PROFILE AND OUTCOME OF CEREBRAL VENOUS SINUS THROMBOSIS"** is a bonafide original work done by **Dr. G.ARAVINDAN** in partial fulfillment of the requirements for **M.D. GENERAL MEDICINE BRANCH-I** examination of the Tamil Nadu Dr. M.G.R Medical University to be held in April 2019, under my guidance and supervision in 2018

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I hereby solemnly declare that the dissertation entitled **"A HOSPITAL BASED STUDY ON CLINICAL PROFILE AND OUTCOME OF CEREBRAL VENOUS SINUS THROMBOSIS"** is done by me at Institute of Internal Medicine, Madras Medical College & Rajiv Gandhi Government General Hospital, Chennai during 2017 under the guidance and supervision of **DR.R.PENCHALAIAH.,M.D.,** This dissertation is submitted to The Tamil Nadu Dr. M.G.R Medical University, Chennai towards the partial fulfilment of requirement for the award of M.D. Degree in General Medicine (Branch I)

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CONTENTS

LIST OF ABBREVIATIONS

ADC	-	APPARENT DIFFUSION COEFFICIENT
AED	-	ANTI EPILEPTIC DRUG
ANA	-	ANTINUCLEAR ANTIBODIES
APLA	-	ANTIPHOSPHOLIPID ANTIBODIES
CSF	-	CEREBROSPINAL FLUID
CT	-	COMPUTED TOMOGRAPHY
CVT	-	CEREBRAL VENOUS SINUS THROMBOSIS
DWI	-	DIFFUSION WEIGHTED IMAGING
FZ	-	FOCAL SEIZURES
EGG	-	ELECTRO ENCEPHALOGRAM
ESR	-	ERYTHROCYTE SEDIMENTION RATE
GCTS	-	GENERALIZED TONIC CLONIC SEIZURE
HB	-	HEMOGLOBIIN
ICP	-	INTRACRANIAL PRESSURE
INR	-	INTERNATIONAL NORMALIZED RATIO
IV	-	INTRAVENOUS ROUTE
LP	-	LUMBAR PUNCTURE
LS	-	LATERAL SINUS

LMWH	-	LOW MOLECULAR WEIGHT HEPARIN
MRI	-	MAGNETIC RESONANCE IMAGING
MRA	-	MAGNETIC RESONANCE ARTEROGRAPHY
MRV	-	MAGNETIC RESONANCE VENOGRAPHY
PT	-	PROTHORMBIN TIME
pts	-	patients
PLED	-	PERIODIC LATERALISED EPILEPTIC DISCHARGES
TS	-	TRANSVERSE SINUS
T1WI	-	T1 WEIGHTED IMAGE OF MRI
T2WI	-	T2 WEIGHTED IMAGE OF MRI
SS	-	SIGMOID SINUS
STS	-	STRAIGHT SINUS
SSS	-	SUPERIOR SAGITAL SINUS
SZ	-	SEIZURES
SC	-	SUBCUTANEOUS ROUTE
UFH	-	UNFRACTIONED HEPARIN

INTRODUCTION

INTRODUCTION

Cerebral venous sinus Thrombosis (CVT) has been recognized since the early 19th century[1] but still remains a diagnostic and therapeutic challenge. Cerebral vein and sinus thrombosis is rare compared to arterial stroke often occurs in young individuals [2]. CVT may occur at any time from infancy to old age most reported cases were women in association with puerperium[3]. Onset of symptoms may be acute sub acute or chronic[4]. Cerebral venous infarction is the most serious consequence of cerebral venous thrombosis venous infarctions are often multifocal bilateral affecting both grey matter and sub cortical white matter

Patient of CVT usually presents with headache, seizure, papilloedema, altered sensorium and focal deficits due to thrombosis of intracranial veins and sinuses resulting in hemorrhagic infarctions and raised intracranial tenstion[2]. The above features are present in various combinations ranging from syndrome of raised intracranial pressure without localization to deep altered sensorium and dense hemiparesis. CVT forms a distinct subgroup of cerebrovascular disease in India and is a leading cause of mortality in women of reproductive age group[3]. In India, most of the cases are seen in post partum period in women, while alcoholism is a significant risk factor in males. Pangayara reported from India that CVT accounted for half of young stroke and 40% for stroke in woman.

Cross et al[5] noted: “Usually recovery is rapid and complete if patient survives the acute episode”. Three fourth of cases of CVT in pregnancy and

puerperium reported by him, survived with good recovery. However, in pre imaging era CVT had been diagnosed exclusively at autopsy and therefore thought to be always lethal. After introduction of heparin in treatment of CVT mortality has come down significantly and most of the recent studies[6,7] reporting mortality < 20% compared to earlier studies reporting mortality between 30-50%. However outcome of CVT is highly unpredictable and it is not unusual to see dramatic recovery in deeply comatose patient and sudden worsening in conscious patients due to extension of thrombosis. With the advent of imaging modalities like CT scan and recently Magnetic Resonance Imaging (MRI) and Magnetic resonance venography (MRV), the diagnosis of CVT has improved significantly. Due to multifactorial causation of this condition, it will be interesting to know whether different pathophysiological mechanisms are operating in different clinical settings.

The ability to accurately detect less clinically severe cases of CVT has modified the “natural history” of this disorder. Thus, in contemporary series, the reported mortality rate ranges between 8% and 14%, Ferro JM in contrast to prior studies within which cause-specific mortality was as high as 30% to 50%. Although some patients with CVT present with catastrophic complications, such as a stroke syndrome with focal neurologic signs or coma, many present with mild or nonspecific symptoms, such as isolated intracranial hypertension, presenting with headache and papilloedema.[2,4] However, conversely to arterial stroke, scarce information exists on natural history and long-term prognosis of CVT.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

Cerebral venous thrombosis or sino-venous thrombosis, as the name implies is a condition which involves cerebral venous sinuses and veins together or independent of each other with thrombotic event of varied temporal evolution. The clinical presentation is varied ranging from syndrome of raised ICT without localization to seizures, focal deficits and deep altered sensorium [2,3.] Some patients may even present as behavioral disturbances as the predominant clinical manifestation, confusing the picture with post partum Psychosis. Strokes resulting from cerebral venous thrombosis usually affect young persons, particularly women in reproductive age group, and carry a high mortality if not managed adequately[10]. The term Primary or Idiopathic Cerebral Venous thrombosis is used when no specific etiological factor is evident. 'Secondary' sino-venous thrombosis results from a variety of causes that include injury, infection, hematological disturbances, dehydration etc[11].

HISTORICAL BACKGROUND

The wide spectrum of clinical features in cerebral venous thrombosis, the varied and changing etiological factors and the apparent “rarity” of the condition had made advances in knowledge slow and uneven. Periods of relative neglect has been interspersed with burst of enthusiastic discussion. The earliest reference to cerebral venous sinus thrombosis was that of Ribes in 1824[1]. He described in detail the clinical and post mortem findings of 45 yr old man who had thrombosis of superior sagittal and lateral sinuses, subdural effusion and metastatic carcinoma in the brain. The first case of puerperal venous thrombosis was reported by John Abercrombie in 1828. His patient, a 24 year old woman, developed headache, delirium and initially right sided than generalized seizures at the beginning of second week after delivery. Autopsy showed ischemic and hemorrhagic infarcts with thrombosed and sclerosed cortical veins. Quinke and Nonne identified the clinical syndrome of pseudo tumor cerebri (a term coined by latter) as a clinical counterpart to sinus thrombosis. Kalbag and Woolf, Sir Charles Symonds and others gave a precise clinical description of CVT after 1940. After introduction of CT scan and recently used MRI with MRV diagnosis of CVT has become simpler as these imaging modalities are quite sensitive in detecting CVT. Several large series with confirmation of diagnosis by angiograms, surgical exploration, and autopsy and recently with CT and MRI studies have been reported from Indian subcontinent,[3,8]

EPIDEMIOLOGY

The true incidence of CVT is unknown. Ehlers and Courville found only 16 superior sagittal sinus thrombosis in a series of 12,500 autopsies (0.12%) [18]. Towbin found CVT in 9% of 182 consecutive autopsies[19]. However, with the more recent reports of large clinical series, the true incidence of CVT is probably considerably higher than that derived from autopsy series. Exact figures however remain elusive. People of all age groups may be affected by CVT but there is preponderance in young women because of specific causes like use of oral contraceptives, pregnancy and puerperium. Puerperal CVT has been reported to account for upto 15-20% of 'young stroke". It is the commonest cause of stroke in young women in India. 50% of strokes in Indian women are related to pregnancy and puerperium and 95.5% of these are due to CVT[5]. In Western countries, the incidence of CVT related to pregnancy and puerperium ranges from 1 in 1666-10,000 pregnancies. Risk factors like hyper homocystenemia**,** OCP use, alcoholism, procoagulant state are increasingly recognized in addition to the conventional risk factors like postpartum state.

RELEVANT VENOUS ANATOMY

The cerebral venous system comprises of cerebral veins that empty into dural sinuses which then drain the blood into two internal jugular veins. Embryologically, the entire cephalic drainage may be subdivided into an outer and superficial segment, which drains the scalp, underlying muscle, and tendons; and intermediate segment which drains the skull, diploe and dura matter; and a cerebral segment, consisting of the veins that drain the brain. The cerebral segment may be further subdivided into superficial cerebral group of veins and deep cerebral group of veins.

The superficial cerebral veins coalesce on the Pial surface draining out the blood from the outer 1 or 2cm. of cortex and the underlying white matter. Venous blood in these vessels travels in a centrifugal direction and ultimately terminates in one of the dural sinuses. The deep cerebral veins serve to drain blood in a centripetal direction away from deep white matter, the basal ganglia, and the diencephalons. Tributaries draining many of the deep structure of the cerebrum join veins in the lateral angles of the ventricles and form a sub ependymal plexus. The veins of this plexus empty into the internal cerebral veins, which join the great cerebral vein of Galen.

Superior Sagittal Sinus (SSS)

SSS lies in the attached border of the falx cerebri and runs from the foramen caecum to the occipital protuberance, where it joins straight sinus, lateral sinus and torcular herophili i.e. confluence of sinuses. The anterior part is narrow or sometimes absent or replaced by two superior cerebral veins that join behind the coronal suture, consequently anterior part of sinus is often poorly visualized on angiography and its isolated lack of filling is not sufficient to indicate thrombosis. The SSS receives superficial cerebral veins and drains major part of cortex. It also receives diploic veins, themselves connected to scalp veins by emissary veins, which explains the incidence of SSS thrombosis after cutaneous infections and contusions SSS and other sinuses play a major role in CSF circulation because they contain most of the arachnoid villi and granulations in which much of the CSF absorption takes place. Thus, there is a direct dependency of CSF pressure accounting for the frequency of raised intracranial pressure, in SSS or Lateral sinus (LS) thrombosis.

Inferior sagittal sinus

Orginates from posterior 2/3 of falx cerebri and joins the great cerebral vein to form the straight sinus that drains into left transverse sinus.

Lateral Sinus

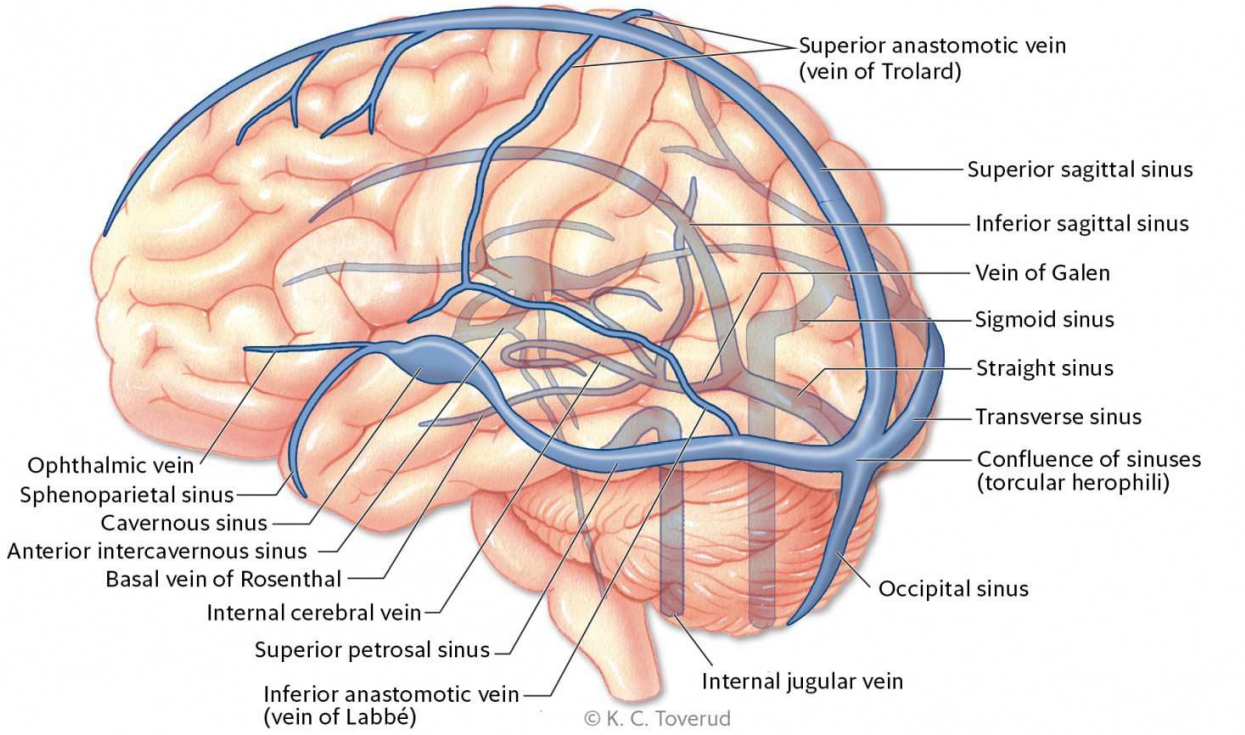
The lateral sinus extends from the torcular herophili to the jugular bulb and consists of the transverse and sigmoid portions. They drain blood from cerebellum, brain stem and posterior portion of cerebral hemispheres. They also receive some veins from middle ear, another possible source of septic thrombosis. Numerous LS anatomic variations may be misinterpreted in sinus occlusions on angiography. The right LS was more often the direct continuation of the SSS and are frequently larger than the left LS which receives most of its supply from straight sinus. The sinus on one side can be poorly developed or even absent.

Sigmoid sinus (SS):

It orginates as continuation of transverse sinus and is ‘s’ shaped, from the postero inferior angle of the parietal bone to the posterior part of the jugular foramen and ends in the superior bulb of internal jugular vein. Its tributaries are the mastoid veins, cerebellar veins, internal auditory vein.

Straight sinus (STS):

It orginates at the junction of the falx and the tentorium and the torcular herophilius where the straight sinus, transverse sinus and SSS meet. In Hacker’s study, transverse portions were not visualized on ipsilateral carotid Angiogram in 14% of cases on left side and 33% on right side, whereas sigmoid portions, which may be directly injected via cerebral veins, failed to fill in 4% of cases on left side and were always demonstrated on right. An isolated lack of filling of a left transverse sinus is more suggestive of hypoplasia than of thrombosis.



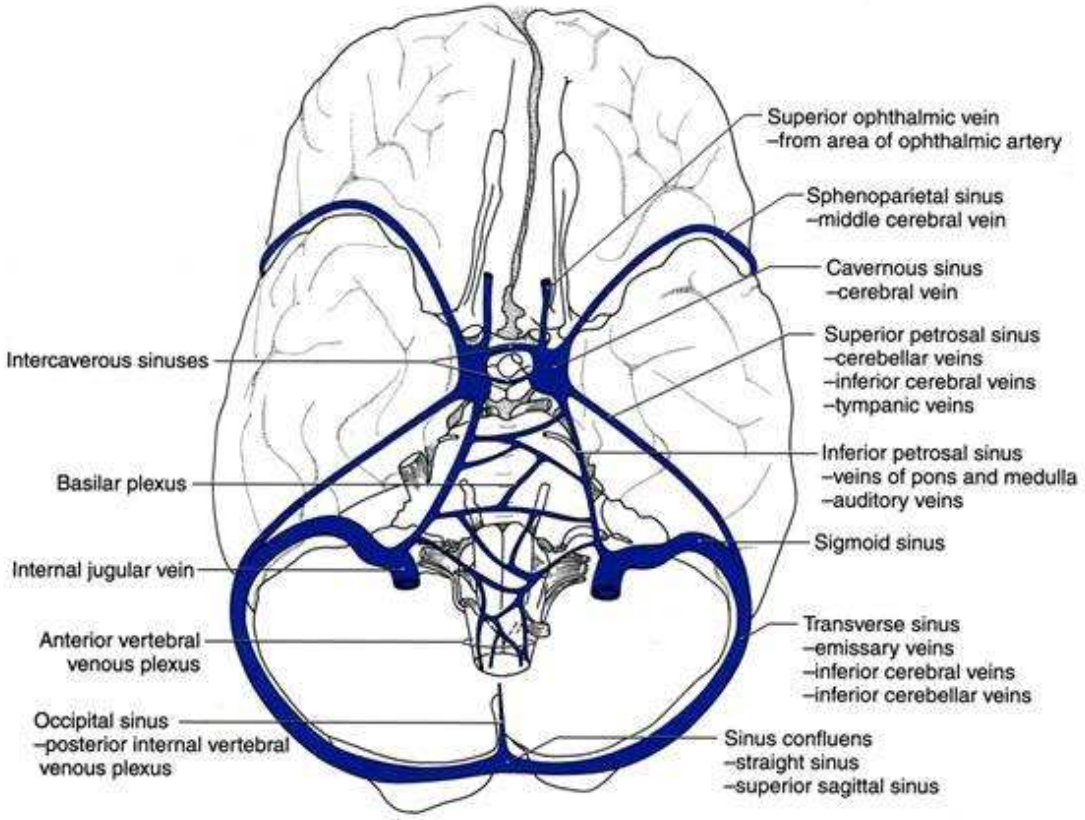
Cavernous Sinus

This sinus drains venous blood from the orbits through the ophthalamic veins and from anterior part of base of brain via the sphinopalatine sinus and middle cerebral veins. They empty into both superior and interior petrosal sinuses and ultimately into internal jugular veins. Because of their situation, cavernous sinuses are often thrombosed in relation to infections of face or sphenoid sinusitis. In contrast to other sites, infection is the leading cause of cavernous sinus thrombosis.

Petrosal and sphenoparietal sinuses

Originates at the junction of tentorium and the petrous bone and it drains into transverse sinuses. The inferior petrosal sinuses lie in between the clivus and petrous ape. It runs medial to the superior sinus joining the jugular bulb.

The sphenoparietal sinus is a medial extension of the sylvian vein and courses around the greater sphenoidwing



They can be roughly divided into 3 groups:

1. Superficial cerebral veins
2. Deep cerebral veins
3. Veins of posterior fossa

Superficial cerebral veins: Some of the cortical veins- the frontal, parietal, occipital and superior cerebral veins drain the cortex ascending to SSS whereas others, mainly the middle cerebral veins drain into the cavernous sinus. Trolard's great anastomotic vein connects the SSS to middle cerebral veins, which are then connected to LS by vein of labbe. The cortical veins

present some peculiarities that are important to know to understand some of the clinical features of CVT. They have thin walls, no muscle fibres and no valves. These features allow for dilatation and reversal of the direction of the blood flow when the sinus into which they drain are occluded. They are linked by various anastomoses, allowing development of collateral circulation (angiographically visible as corkscrew vessels). This probably explains the good prognosis of some of the thrombosis.

Deep Cerebral Veins: The internal cerebral and basal veins both join to form great vein of Galen, which continues as straight sinus drain blood from deep white matter of the cerebral hemispheres and from Basal ganglia. In contrast to superficial system, the deep system anatomy is constant and is always visualized on angiography, so that thrombosis is easily recognized.

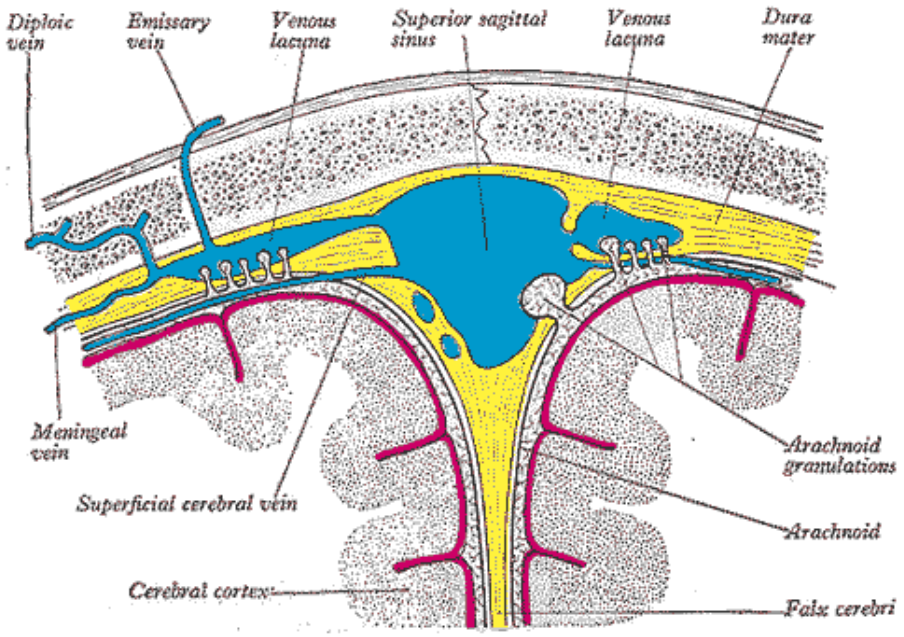
Veins of posterior fossa: There are 3 groups:

1. Superior veins draining into Galenic system
2. Anterior veins drawing petrosal sinuses
3. Posterior veins draining into the torcular or neighboring SS and LS

They are variable in course and angiographic diagnosis of their occlusion is extremely difficult.

Microscopic Anatomy of Cerebral Veins and Sinuses

Capillaries open in to cerebral venules which apart from their wider lumen are indistinguishable from them. These venules join the small medullary or cortical veins. These vessels reach the ventricular or cortical surface, either directly or indirectly, after fusion with neighboring veins forming larger vascular stems. The walls of cerebral veins consist of an endothelial lined tunica intima. Surrounding the endothelium is a thin adventitial layer. Veins do not have clearly defined muscular layer or values. There is little to suggest than the veins receive vasomotor innervations. As these vessels approach their destination they become more fibrous and resemble closely the structure of dural sinus. The strategic location of the sinuses within the major folds or junctions of dura, the firm attachment of the dura to bone, and its tough fibrous consistency maintains patency of the sinuses all the time. The walls of dural venous sinuses consist of an inner lining of endothelium and an outer layer essentially the same as dura elsewhere. The outer layer consists chiefly of fibroblasts and large interlaced bundles of collagenous fibres. A few nerve fibres, presumably afferent have been reported to innervate along the dural venous sinuses. The walls of the venous sinuses are not uniformly smooth and in certain locations like middle third of SSS are thrown into folds or membranous irregularities. According to one hypothesis, these folds may perform valve like action, while other authors suggested that they may be important in maintaining laminar flow.



Warwick R, Williams PL. Gray's Anatomy, 35th British ed. Philadelphia: W.B. Saunders, 1973. fig. 7.166, p. 991.

CAUSES OF CVT

Several medical, surgical and gynaeco-obstetric ailments as well as a number of regional causes like infective, trauma, tumors etc. have been implicated in the causation and predisposition to CVT, Table (1) lists the recognized causes or predisposing conditions[11].

Table 1: Causes of cerebral venous thrombosis

A. <u>SEPTIC DURAL SINUS THROMBOSIS</u>

Local-

Sepsis , Trauma

Intracranial infections:

Abscess, empyema and meningitis, Otitis, Sinusitis, Tonsillitis Stomatitis

Systematic -

Bacterial (Typhoid, TB, Septicemia, Endocarditis)

Viral (Measles virus, Hepatitis viruses, Herpes Simplex virus, HIV, Cytomegalovirus)

Parasitic (Malaria, Trichinosis) Fungal (Aspergillosis)

B. <u>NONSEPTIC DURAL SINUS THROMBOSIS CAUSES:</u>

Altered hemodynamic states

Dehydration

Fever

Cardiac failure

Hematological disorders:

Polycythemia Vera, Secondary polycythemia

Disseminated intravascular coagulation, Sickle cell anemia and trait

Cryoglobulinemia

Paroxysmal nocturnal hemoglobinuria Thrombocytosis

Severe anemia

Antithrombin – III deficiency Protein C & S deficiency

Antiphospholipid antibody syndrome

Hormonal dysfunction

Oral contraceptive use, Pregnancy and puerperium Androgens

Trauma

Penetrating & non-penetrating head injuries

Surgery

Cardiac pacemakers Jugular venous catheters

Metabolic disorders

Homocystinuria

Osteopetrosis Diabetes mellitus

Neoplasia

Meningioma

Metastasis (usually hematogenous)

Inflammatory disorders

Behcet’s disease Sarcoidosis

SLE

Wegener’s granulomatosis Polyarteritisn odosa Inflammatory bowel disease

Ulcerative colitis Crohn’s disease Cogan syndrome

Vascular disorders

Arterio-venous malformation Arterial occlusions

Sturge weber syndrome

CLINICAL PROFILE

The spectrum of symptoms and signs among patients with CVT is remarkably variable. Patients present with varying combination of headache, seizures, aphasia, behavioral abnormality, altered sensorium and deficits. The onset may be acute, sub acute or chronic. The presentation is acute in obstetric and infectious CVT while a slowly progressive disease is more common in inflammatory and Idiopathic cases [7,11]. Bilareral papilloedema and symptoms of raised Intracranial Pressure occur in those with large sinus (SSS and LS) thrombosis blocking CSF absorption. Cortical deficits like agnosia, apraxia, cortical blindness and aphasia do occur but better recognized in mild illness with good sensorium. Some patients may present with psychotic features before manifestations of raised intracranial pressure or focal deficits sets in. Monoplegia (brachial or crural), hemiparesis with leg more affected than arm, intact language despite right hemiparesis are all common but generally regress without residual deficits. Cerebellar infarcts with edema acting like space occupying lesion, requiring surgical decompression are rarely encountered.

HEADACHE:

The most prominent and frequently presenting symptom is the headache. Most of the time, it is only complaint. About 70-80% of the patients presents with headache.[35] there are no specific features. It may be acute, like a thunderclap headache, [36] or sub acute or chronic. Sometimes migrane like headache also presents associated with nausea, vomiting and other neurological symptoms. The cause may be due to spillover blood stimulates the pain-sensitive fibers in the Dura or due to rise of intracranial pressure[37].

Sometimes, the only manifestation of CVT is an isolated headache[35] and venogram shows involvement of lateral sinus thrombosis. Isolated headaches were usually unilateral and ipsilateral to the involvement of lateral sinus. And as the other clinical features of CVT , headache also reduced or cured by treatment with heparin.

SEIZURES

More than arterial stroke, seizures common in CVT. Seizures begins early, may be focal or generalized. Among CVT, seizures present in 50% of cases. Seizures may be focal, modal or generalized. Early appearance of seizure and Status epilepticus were sign of bad prognosis[45],may be followed by aphasia and hemiparesis. It is commonly due to 'irritation' of the cerebral cortex either directly or due to blood spill. seizure incidence varies from as low as 3.5% to as high as 42% in two Indian series.[47,48]

FEVER

More often seen in patients, due to Sepsis or secondary to systemic infections. It is frequently seen in Indian patients , but it is not mentioned as an important feature in two large international Studies[41, 42]. Fever does not necessarily imply Infection and is probably caused by an aseptic thrombotic process.[43, 44] Superior sagittal sinus (SSS) thrombosis in infants and children may present only with fever and convulsions, important differential diagnosis for febrile seizures.

ALTERED SENSORIUM

Altered sensorium is observed in 20–30.6% of patients with CVT. Sassi et al. (2016) conducted a retrospective study in which 49 of 190 CVT patients had altered consciousness. Of the patients with altered consciousness, 28.6% (14/49) had mild altered consciousness (GCS10-15), 63.3% (31/49) had moderate altered consciousness (GCS8-10 points), and only a very small number of patients (4/49) had severely altered consciousness (GCS 3-7).

Patients with deep venous systemthrombosis often experience altered consciousness) Bousser and Ferro, 2007). Terazzi et al. (2005) found that 61.5% of patients with deep vein CVT but only 17.1% of patients with cortical CVT had altered consciousness or confusion. Pfefferkorn et al. (2009) conducted a retrospective case study of 32 patients with deep venous thrombosis. In these Patients, headache (81%) and a decreased level of consciousness (72%) were the most common symptoms, and they were usually

accompanied by focal neurological deficits and neuropsychiatric Manifestations, such as confusion and amnesia. Of the 32 included patients, 72% (23 cases) had a GCS -14, whereas 38% (12 cases) had a GCS -8. Four patients had a “hyperacute” course, meaning they had a GCS score - 8 within 24 h of onset.

FOCAL NEUROLOGICAL DEFICITS

Some CVT patients present with neurological deficits, including motor/sensory impairment, aphasia, cranial nerve palsy, and cortical blindness, as the main manifestation. Neurological focal lesions are associated with larger cerebral infarctions involving the proximal midline of the Rolandic region, the posterior temporal region, the frontal-parietal and the parietaltemporal region. Focal neurological deficits are more common in non-inflammatory CVT, and cavernous sinus syndrome is more common in infection-related CVT (Paciaroni et al., 2008; Korathanakhun et al., 2015).

Motor deficits were the most common neurological deficits and were observed in 19.1–39% of CVT patients. Motor deficits were more common in CVT patients with superior sagittal sinus, cortical veins, and cerebral deep venous system involvement. Studies have shown that almost the same proportion of left and right limb involvement is observed. Motor deficits in CVT can be similar to those in arterial stroke, including a sudden onset, and they can also be similar to those in an intracranial spaceoccupying lesion with subacute onset. A very small number of patients can manifest symptoms like those observed in a transient ischemic attack (TIA; Ferro et al., 2004; Bousser

and Ferro, 2007; Paciaroni et al., 2008; Wasay et al., 2008; Uzar et al.,2012).

Aphasia is also a common neurological defect that is observed in 19–24% of CVT patients. Aphasia is commonly observed in left lateral sinus thrombosis or deep vein thrombosis (Bousser and Ferro, 2007).

PAPPILEDEMA

Dural venous sinuses thrombosis leads to an increase in venous pressure. This alters the intra cranial pressure gradient leading to poor CSF absorption through the pacchionian granulations, which cause the rise of ICP and thus pappiledema. The incidence of pappiledema is variable. It depends on the aetiology of CVT and site of venous thrombosis. One observation finding is papilledema is less common in puerperal CVT. In Indian patients, incidence of papilledema varies from 7.4-55%.[34, 36, 50] Idiopathic intracranial hypertension can be as high as 83-100%.[13, 36] among the subacute onset of clinical presentation of CVT.

By the clinical presentation, patients were categorized as 4 groups

Group-I: Meningo encephalitic type

headache, fever, seizures,altered sensorium, focal deficits, meningeal signs.

Group-II: Acute fulminant type :

Status epilepticus, coma.

Group-III: Pseudo tumour type :

Headache ,vomiting , pappiledema

Group-IV: Neuropsychiatric type :

Abnormal behaviour, with or without features of raised intracranial tension.

Clinical features of cerebral venous thrombosis in various Indian series

CLINICAL FEATURES	BANSAL[13] n=138(%)	SRINIVASAN[26] n=135(%)	NAGARAJA[3] n=405(%)
1. Fever	62	16	
2. Headache	48	24	70.8
3. Vomiting	36	24	38
4. Seizures			
i.GTCS	29	50	39.7
ii.Focal	17	22	30.1
5. Dysphasia	25	5	-
6. Diplopia	1	-	-
7. Nuchal rigidity	3	10	13
8. Deep leg vein thrombosis	10	-	-
9. Altered sensorium	41	43	58
10. Papillodema	35	16	18.5
11. Ocular palsy	-	2	11
12. Motor deficit	69	49	66.4

Clinical presentation in the two largest series of CVT patients

Ferro et al.: 624 patients	
Headache	88.8%
Visual loss	13.2%
Papilledema	28.3%
Diplopia	13.5%
Stupor or coma	13.9%
Aphasia	19.1%
Mental status disorders	22%
Any paresis	37.2%
Any seizure	39.3%
Sensory symptoms	5.4%
Other focal cortical sign	3.4%
Wasay et al.: 182 patients	
Headache	71%
Generalized weakness	54%
Focal motor or sensory deficit	36%
Nauese/vomiting	35%
Seizures	32%
Walking difficulty	30%
Drowsiness	28%
Visual blurring	23%
Dizziness	21%
Behavioural symptoms	18%
Slurred speech/inability to speak	16%
Coma	15%
Fever	14%

Nagaraja et al[3] grouped clinical features of CVT in four categories depending upon the topographical venous involvement.

1. Presentation with seizures, focal deficits and progressively deteriorating consciousness. Thrombosis involves the dural sinuses as well as cortical veins producing cerebral infarction. Seizures may be focal, multi focal or generalized. Paralysis may be unilateral or bilateral and is usually maximal in lower limbs. Later during the course, patient may manifest signs of tentorial or central herniation leading to coma and death.

2. Presentation with symptoms and sings of raised intracranial tension namely headache, vomiting and papilloedema. If thrombosis continues to dural sinuses, the course is usually slow and prognosis is favourable.

3. Occasionally, thrombosis predominantly involves cortical veins and patient may present with feature of space occupying lesion.

4. Rarely, thrombosis predominantly involves the deep venous system. Patient manifest symptoms of raised intracranial tension, focal deficits, choreoathetosis, ocular sings and coma. It runs a fulminant course Cavernous sinus thrombosis is usually due to spread of infection from face, para nasal sinus or intracranial venous sinuses. It has a distinctive clinical picture where patient presents with fever, chills, toxemia with proptosis, chemosis and painful ophthalomoplegia, initially unilateral but often becoming bilateral. Papilloedema and retinal haemorrhages indicate retinal vein thrombosis

CLINICAL FEATURES DESCRIBED BY AMERI ET AL[18] 1992

Headache	83(75%)
Papilloedema	54(49%)
Motor or sensory deficit	38(34%)
Seizures	41(37%)
Drowsiness, mental changes, confusion or coma	33(30%)
Dysphasia	13(12%)
Multiple cranial nerve palsies	13(12%)
Nystagmus	2(2%)
Hearing loss	2(2%)
Bilateral or alternating cortical signs	3(3%)
Cerebellar incordination	3(3%)

In another series Cantu et al[7] from Mexico analyzed clinical radiological features of 113 cases of CVT comparing obstetrical cause related CVT to non-obstetrical cause related CVT. Clinical features in this series was similar to other series but in “obstetrical group” symptoms evolved more rapidly and the outcome in terms of mortality was less.

Investigations

Computed tomography scan with contrast injection is the first neuroimaging examination to be carried out when CVT is suspected as it is easily available and has good sensitivity and specificity [2,5,7,11,19,21]. CT Scan is usually abnormal in 80-85% of cases of CVT. Normal scans are particularly common early in the course. CT sings of sinus thrombosis may be focal or generalized and may result from the thrombus itself or from its sequelae. They are seen before or after the infusion of the contrast.

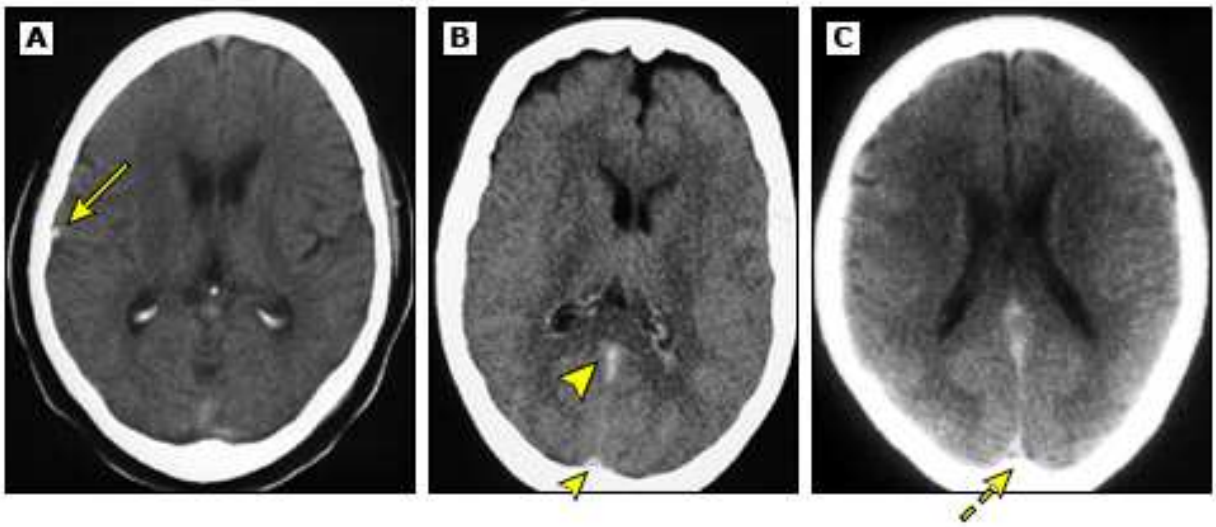
CT SCAN:

On plain CT Scan, the thrombosed superior sagital sinus may appear as an unusually dense triangle which is sometimes referred as “dense” or “filled delta sign”. The straight sinus and vein of Galen may also appear hyperdense before contrast when they are thrombosed.

The “cord sign” which is considered pathognomic of cortical venous thrombosis, is a round hyperdensity seen on several sequential slices due to presence of thrombus in the lumen of a vein.

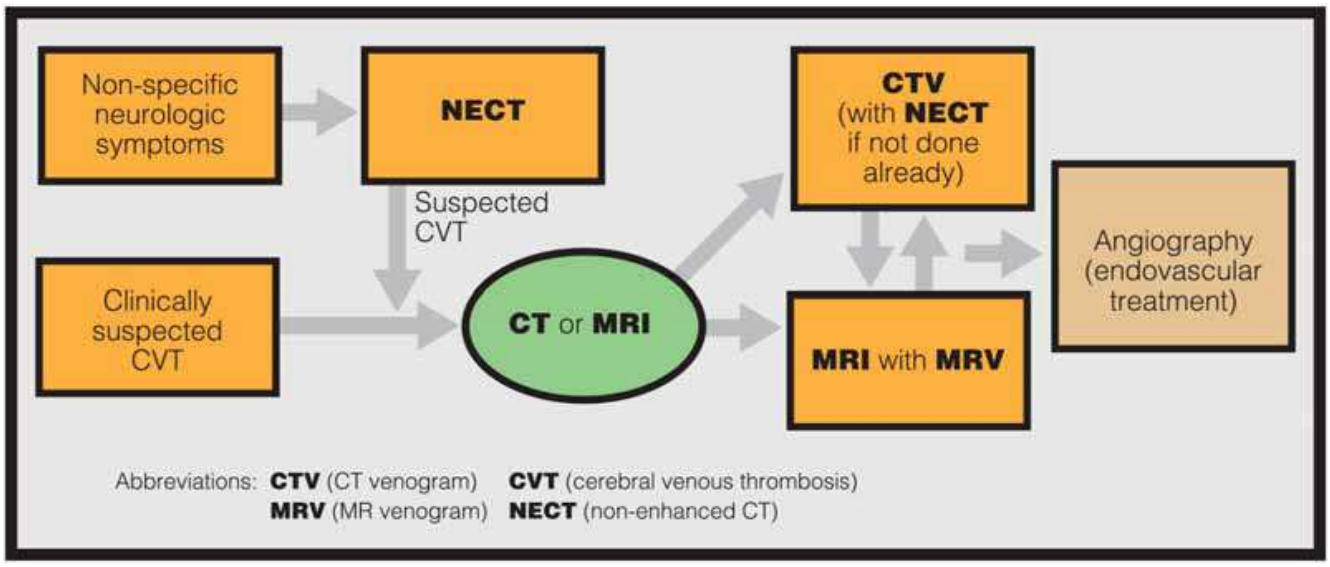
Intraparenchymal linear hyperdensities representing thrombosed intracerebral veins have the same significance.

Areas of Ischemia may be imaged as mixed density lesion representing haemorrhagic infarction. Haematoma may also be seen. The location of these lesions correlates poorly with the site of occluded vein. Evidence of increased intracranial pressure such as focal edema and compression of the ventricles, subarachnoid spaces and cisterns may also be reliably imaged on CT scan.



A) Noncontrast head CT shows a hyperdense thrombosed cortical vein (arrow).
B) Noncontrast head CT shows a hyperdensity in the torcula (small arrowhead) and the straight sinus (large arrowhead), a direct sign of dural sinus thrombosis (**the dense triangle sign**).
C) Head CT shows non-filling of the confluent sinus after contrast injection (**the empty delta sign**).

One of the best known and most specific sign seen after contrast is "empty delta sign"[20]. This consists of a central lucency within the superior sagittal or straight sinus, which is surrounded by a margin of contrast enhancement. In aggregate data, 20-30% of cases show the empty delta sign but it is usually not seen for 3-4 days after the occlusion.[27]



ALGORITHUM FOR IMAGING IN CVT CASES.

RADIOLOGICAL FINDINGS IN A SERIES OF 113 PATIENTS BY CANTU ET AL7 1993

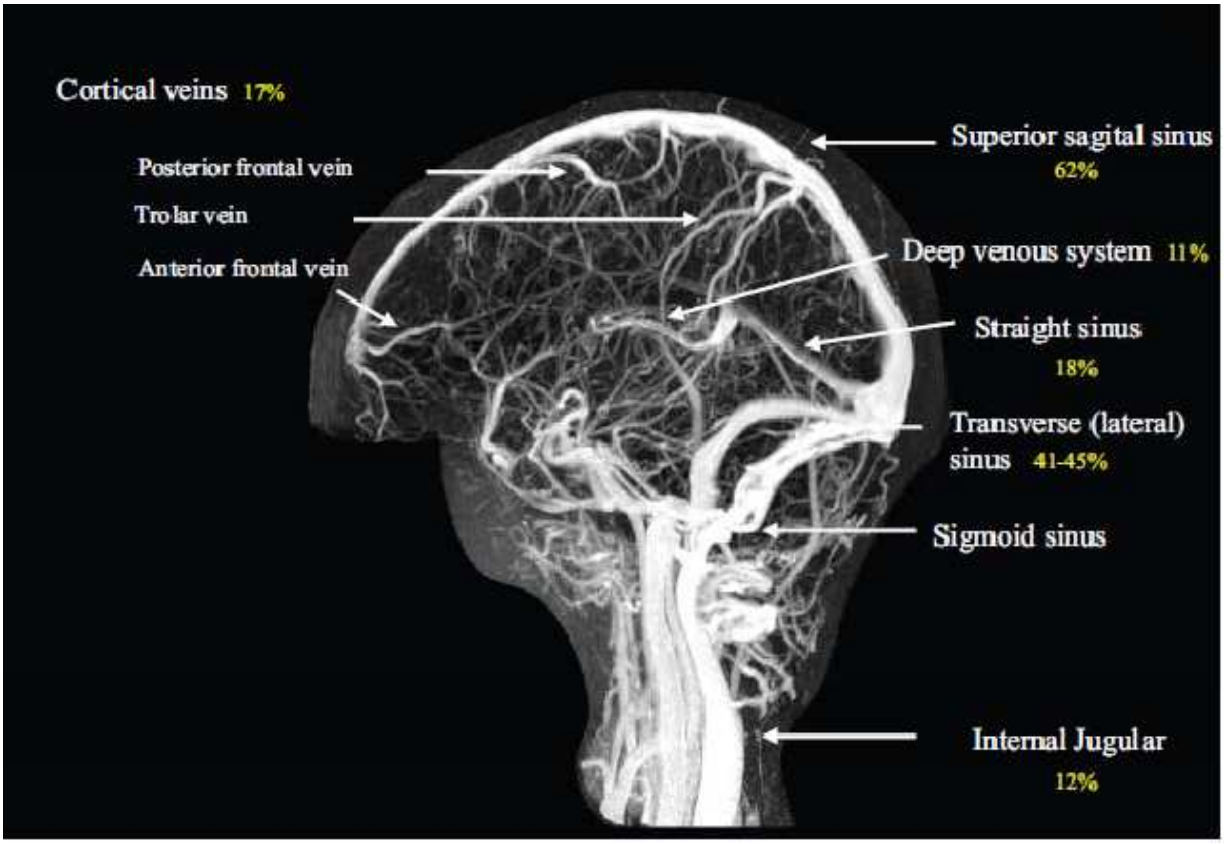
* delta sign, dense triangle or empty delta sign

Findings	uted Tomographic Scan		gnetic Resonance Imaging	
	Puerperal	Non-puerperal	Puerperal	Non-puerperal
Normal	5 (8.4%)	4(11.1%)	0 (0%)	0 (0%)
Signs of CVT*	19 (32.2%)	13 (36.1%)	17 (89.4%)	19 (95%)
Non-haemorrhagic venous infarct	16 (27.1%)	7(19.4%)	3 (15.7%)	2 (10%)
Haemorrhagic venous infarct	21(35.5%)	12(33.3%)	10 (52.6%)	11 (55%)
Intracerebral haemorrhage	6 (10.1%)	5 (13.8%)	2 (10.5%)	4 (20%)
Unilateral lesions	25 (42.3%)	15 (41.6%)	8 (42.1%)	12 (60%)
Bilateral lesions	18(30.5%)	9 (25%)	7 (36.8%)	5 (25%)

MRV

Four vessel Angiography (conventional or digital subtraction angiography) with visualization of the entire venous phase on at least two projections (frontal and lateral views) best visualize the SSS and other sinuses. This can be especially useful if clinical history is not available and CT Scan is showing mixed density lesion mimicking both contusion and CVT. After the advent of MRI and MRV use of conventional angiography has become limited as MRV is very sensitive and non invasive method of diagnosing CVT [8,9,10,21]. MRI and MRV are particularly useful in the setting where patient presents with syndrome of raised intracranial tension without localization and normal CT Scan. Here sinus thrombosis can be demonstrated by MRV. Murthy et al (1990) presented findings of MRI in 16 cases of CVT Evidence of venous sinus thrombosis alone was seen in three cases, only haemorrhagic venous infarct in two cases and a combination of both in 11 cases. In five patients where CT showed delta sign MRI showed hypo intense signals in the centre of superior sagittal sinus with a rim of surrounding hyper intense in T1 as well as T2 sequences. SSS is the most common sinus affected followed by lateral sinus. Superficial venous system is more commonly affected f/b the deep venous system. For chronic CVT due to isolated cortical vein involvement gradient (swi image) can be used

MRV IMAGE SHOWING CEREBRAL VEINS AND SINUS



LABORATORY TESTS

Aside from neuroimaging, there is no simple confirmatory laboratory test that can confidently rule out CVT in the acute phase of the disease. Guidelines from the American Heart Association/American Stroke Association (AHA/ASA) recommend obtaining routine blood studies consisting of a complete blood count, chemistry panel, prothrombin time, and activated partial thromboplastin time for patients with suspected CVT[1]. The findings from these tests may suggest the presence of conditions that contribute to the development of CVT such as an underlying hypercoagulable state, infection, or inflammatory process. The guidelines recommend screening for these and other potential prothrombotic conditions that may predispose to CVT, including use of contraceptives, at the initial clinical presentation.

D-dimer:

An elevated plasma D-dimer level supports the diagnosis of CVT, but a normal D-dimer does not exclude the diagnosis in patients with suggestive symptoms and predisposing factors. A 2012 meta-analysis included 14 studies that evaluated D-dimer in 1134 patients for the diagnosis of suspected or confirmed CVT. In seven studies that evaluated patients with suspected CVT, D-dimer was elevated in 145 of 155 patients in whom CVT was confirmed, and was normal in 692 of 771 patients in whom CVT was ruled out, yielding a sensitivity and specificity of 94 and 90 percent, respectively. The sensitivity of D-dimer for CVT was also lower in patients with isolated headache as the

presenting symptom (82 percent), in those with sub acute or chronic clinical presentations of CVT (83 percent), and in those with a single affected venous sinus (84 percent).In a subsequent study of 233 patients with suspected CVT and symptom onset of less than seven days, D-dimer demonstrated a sensitivity and specificity of 94 and 98 percent, respectively, for predicting CVT.

Lumbar puncture:

Lumbar puncture may be useful to exclude meningitis in patients with CVT who present with isolated intracranial hypertension, a syndrome that may account for up to 25 percent of all patients with CVT[5]. In addition, lumbar puncture is valuable in such patients to measure and decrease cerebrospinal fluid pressure when vision is threatened. However, in the absence of suspicion for meningitis, cerebrospinal fluid analysis is usually not helpful diagnostically for patients with focal neurologic findings and neuroimaging confirmation of CVT [1].

The cerebrospinal fluid abnormalities in CVT are nonspecific and may include a lymphocytic pleocytosis, elevated red blood cell count, and elevated protein; these abnormalities are present in 30 to 50 percent of patients with CVT [41,42]. Performing a lumbar puncture is not harmful in patients with CVT, as suggested by the findings of a study that analyzed 624 patients with CVT and identified 224 who had lumbar puncture [44]. The groups with and without lumbar puncture did not differ on any of the outcome measures. However,

lumbar puncture is contraindicated in patients with large brain lesions because they have an increased risk of herniation.

EVALUATION FOR THROMBOPHILIC STATE:

Searching for a thrombophilic state, either genetic or acquired should be done for patients with CVT who have a high pretest probability of severe thrombophilia, a category that includes those with a personal and/or family history of venous thrombosis, CVT at a young age, and CVT in the absence of a transient or permanent risk factor [1,2,3,5]. When appropriate, screening should include:

●Antithrombin

●Protein C

●Protein S

●Factor V Leiden

●Prothrombin G20210A mutation

●Lupus anticoagulant, anticardiolipin, and anti-beta2 glycoprotein-I antibodies

Acute thrombosis can transiently reduce levels of antithrombin, protein C, and protein S, so the utility of testing for these disorders in the acute phase of CVT is limited. In practice, it is preferable to test for protein C, protein S, and antithrombin at least two weeks after oral anticoagulation has been discontinued, since warfarin therapy reduces measurements of protein C and protein S, and may raise plasma antithrombin concentrations into the normal range in patients with hereditary antithrombin deficiency. It is possible to test

for protein C and protein S levels while receiving heparin therapy, which does not alter plasma protein C or protein S concentrations. However, testing for antithrombin should be performed when off heparin, which can lower antithrombin levels.

No underlying etiology or risk factor for CVT is found in approximately 13 % of adult patients in recent studies. However, it is important to continue searching for a cause even after the acute phase of CVT, as some patients may have a condition such as the antiphospholipid syndrome, polycythemia, thrombocythemia, malignancy, or inflammatory bowel disease that is discovered weeks or months after the acute phase.

If abnormal results are found in assays for lupus anticoagulant, anticardiolipin, or anti-beta2 glycoprotein-I antibodies, testing should be repeated at least 12 weeks later, as the diagnosis of antiphospholipid syndrome requires two positive determinations of these biomarkers.

An evaluation for paroxysmal nocturnal hemoglobinuria should be pursued if the complete blood count shows unexplained hemolytic anemia, iron deficiency, or pancytopenia.

In patients older than 40 years without identified etiology, we suggest searching for an occult malignancy. In patients with sepsis, or with fever and no obvious cause of infection, we recommend performing a lumbar puncture

PATHOGENESIS OF CVT

Various theories have been put forward regarding pathogenesis of CVT, particularly in relation to puerperal CVT. Some of them are

1. Infective theory
2. Matin – Baston theory of embolic – thrombus
3. Kendall's theory of local damage
4. Statis theory
5. Hypercoaguable state theory

INFECTIVE THEORY

The septic thrombosis due to infection occurs with attendant Thrombophlebitis. The classic examples of such a condition are cavernous sinus thrombosis because of facial infection and lateral sinus thrombosis following otitis media. In Daif a et al.[21] 40 cases of CVT was studied, it showed infection was the cause of CVT in 7% of cases, whereas infection was the cause in 16% and 17% of cases reported by Bousser et al[6] ,Shell and Rathe, [22] respectively.

MARTIN-BATSON THEORY OF EMBOLIC THROMBOSIS:

In understanding the pathogenesis of puerperal CVT studies of Batson (1940), and extension of the results of the study by Martin (1941) are milestones. Batson in experimental work on monkeys and human cadavers

showed that pelvic veins have anastomosis with cerebral plexus of veins. Though he demonstrated anatomical connection in human cadavers, positive proof of functional conduct in live patients has not been shown. Based on this data Martin argued that thrombi from pelvis of parturient women under circumstances of raised intra-abdominal pressure could pass into vertebral plexus and then to intracranial sinuses. Once the thrombus reaches SSS, where blood flow is slow, it acts as a nidus for further thrombosis. The Martin-Batson theory does not explain the fact that SSS is most frequently involved although the vertebral plexus of veins communicate with the occipital and petrosal sinuses and not SSS. It also fails to explain the delayed onset of symptoms and the basic mechanism of CVT

KENDALL'S THEORY OF LOCAL DAMAGE:

Kendall (1948)[28] put forward his hypothesis of local damage in the sinus. He suggested that damage to the sinus endothelial lining occurs during the periods of breath holding and straining which may occur during the second stage of labor. The opposition to this hypothesis is that while most of the female population becomes pregnant and delivers and many of them repeatedly, less than 0.04% of them develop thrombosis of the SSS.

STASIS THEORY:

Bailey (1971) search stated that CVT involving SSS thrombosis, the oldest thrombus lies in the middle fifth of the sinus and in some cases only the central portion was involved. It suggests the involvement of certain local

factors. The hypothesis is that superior cerebral veins enter the sinus in a direction opposite to the direction of blood flow and there is sudden widening of the sinus at that specific point and this may contribute to the localisation.

THEORIES OF HYPER COAGULABILITY:

Sinclari [29] (1902) was perhaps the first to demonstrate that plasma fibrinogen levels increase up to 150% of normal in the last trimester of pregnancy and attributed it to the increased tendency to thrombosis at this time of puerperium. In addition to humoral factors contributing to hyper coaguable state, Chopra et al [30] (1979) and Bansal et al[13] (1980) have shown that there is increased platelet adhesiveness during pregnancy and puerperium. They demonstrated that peak increase in platelet count occur by 10th postpartum day, when the incidence of CVT is higher. Chopra and Prabhakar[30] (1979) found statistically significant higher levels of beta lipoproteins and triglycerides in 27 patients of CVT compared to 15 controls. Hyper coagulability induced by oral contraceptive has been incriminated to cause CVT in a few reports Gettlefinger[34] 1977). Summarizing hyper coagulability in the form of increased levels of plasma fibrinogen, factor VII and X, decreased fibrinolytic activity, increased platelet count and adhesiveness, and increased phospholipids occur in normal puerperium and may contribute to CVT. Stasis and endothelial damage may also play a role. Thus one or more of the above factors may be responsible for puerperal CVT.

OTHER CAUSES:

In addition to above mentioned abnormalities, other factors held responsible for hypercoaguable state are anemia and dehydration (Kalbag and Woolf [35] 1973; Srinivasan and Natarajan[32] 1979). In Srinivasan's series [12] (1983) 25/135 had less than 9 gm% and in Nagaraja's series [3] (1987), 56% of patients had hemoglobin less than 10 gm%, out of 200 patients of puerperal CVT. Other relatively recently recognized important factors causing hypercoaguble state leading to CVT are factor V leiden mutation, anti-cardiolipin and lupus anticoagulant antibody, protein C and S, and antithrombin III deficiencies[24,31,32]. Deschiens et al [36] (1996) studied coagulation parameters, including activated protein C resistance associated with factor V leiden mutation and anti cardiolipin antibodies, in a series of 40 patients with CVT with or without identified cause or risk factor. 10% had factor V leiden mutation and 8% had increased anticardiolipin antibodies. They suggested that although present in a number of CVT cases these abnormalities are almost invariably associated with other precipitating factors and their presence should not deter the search for other potential cause.

MANAGEMENT

The natural history of CVT is highly variable, from an acute, fulminant course at one end of spectrum to a slowly evolving course without associated neurological deficits at the other. Previously treatment usually comprised of anti-edema measures like IV mannitol and administration of steroids with anticonvulsants. Decompressive craniectomy is also used occasionally. Controversy regarding use of heparin has been resolved more than a decade ago[39] and now heparin is definitely indicated even in precence of haemorrhagic infarction and is safe and effective. However dose and duration of heparin therapy is still to be established. In patients with post partum CVT heparin during acute phase may be sufficient and if needed can be followed by oral anticoagulants for 3 months or more. In patients with deficiency of antithrombin III, protein C, or protein S, prolonged use of anticoagulant is warranted.

ACUTE PHASE

1. Treatment of the Etiology

2. Antithrombotic Treatment

Subcutaneous low-molecular-weight heparin – dose 1mg/kg bd or intravenous unfractioned heparin – dose 5000 IU qid. In experienced centers, if neurologic worsening occurs despite heparin and the best medical treatment and other causes of deterioration are excluded, local intravenous thrombolysis with/without mechanical thrombectomy is an option.

3. Symptomatic Treatment

a. Antiepileptics

In patients with acute seizures and supratentorial lesions.

b. Intracranial hypertension

- Headache – Analgesics
- Lumbar puncture, if there is no parenchymal lesions; perform before starting anticoagulation
- Acetazolamide

c. Impairment of consciousness of herniation

- Osmotic therapy
- Sedation and hyperventilation
- Hemicraniectomy

d. Threatened vision

- Lumbar puncture, if there are no parenchymal lesions; perform before starting anticoagulation
- Acetazolamide
- Lumboperitoneal shunt
- Optic nerve fenestration

POST-ACUTE PHASE

1. Treatment of the Etiology

2. Antithrombotic Treatment

Oral anticoagulants

For 3–6 months if CVT related to a transient risk factor

For 6–12 months if CVT idiopathic or related to “mild” hereditary thrombophilia

For life for recurrent CVT or “severe” hereditary thrombophilia

3. Symptomatic Treatment

Antiepileptics

In patients with acute seizures or seizures in the post-acute phase and supratentorial lesions.

OUTCOME

Before the advent of CT scan and angiography, CVT was diagnosed mainly at autopsy, and so prognosis was considered almost fatal. After the introduction of angiography and before the introduction of CT, the mortality rate varied from 20% to 50%. With the availability of CT and MR imaging as routine investigative tools, milder cases are increasingly recognized, making the outlook more favorable. Overall, mortality varies from 15% to 20%. (Most deaths in CVT are caused by raised intracranial tension and herniation) Although CVT carries a higher mortality than arterial infarction, the morbidity is less. Only a small percentage of patients are left with neurologic sequelae. Recurrence of CVT is not common; when present, symptomatic causes, including deficiency of protein C, protein S, and antithrombin III, should be investigated.

AIM AND OBJECTIVES

1. Identify the etiological spectrum of patients with cerebral vein /sinus thrombosis
2. To attempt correlation between site of venous occlusion and clinical parameters
3. Prognosis of CVT.
4. Factors associated with poor outcome

MATERIALS AND METHODS

MATERIAL AND METHODS

This study was performed as a hospital based retrospective& prospective observational study at MADRAS MEDICAL COLLGE at CHENNAI, TAMILNADU, and India. All the patients admitted in our hospital with the diagnosis of CVT were subjected to neuroimaging techniques, fulfilling the study criteria were recruited by simple random sampling and data collected was analyzed by correlation studies

All patients hospitalized in between the period of 1 year (august 2017 to October 2018) with the final diagnosis of CVT (confirmed by imaging MRI/MRV OR angiography) to be included.

Age – all patients above 13 years of age.

EXCLUSION CRITERIA:

1. Patients who were initially diagnosed as CVT, But MRV/angiogram were normal

2. Patients below 12 years of age.

3. Known case of seizure disorder, migraine, cranial nerve palsies, hemiplegic patients (not able to differentiate the presentation is due to CVT or they already got that complaints)

STUDY DESIGN

Prospective, observational, non interventional clinical study with CT or MRI

STUDY CENTER

Institute of Internal medicine at MADRAS MEDICAL COLLEGE, Chennai

CONSENT:

Patients included in the study after obtaining signed informed consent form after explaining the purpose of the study to the patient or to the patient's attender.

IMAGING, DEMOGRAPHIC AND CLINICAL DATA, RISK FACTORS AND TREATMENT:

A detailed proforma with the following information abstracted and entered into the computerized data sheet, viz; demographic data dates of onset of symptoms, of hospital admission and of confirmation of the diagnosis by imaging symptoms and signs from onset and diagnosis

Mode of onset,-

Acute - 0-48 hr,

Sub acute - 48hours -29 days,

Chronic - >30 days.

Clinical features-

Full and thorough general and physical examination done for all the patients to find out any Focal signs, ICH /Papilloedema signs, Sensorial status. Glasgow coma scale (GCS) scoring was done on admission and during the clinical course;

If patients presenting with seizures history or having seizures, they were grouped as either generalized, partial, complex seizures or status epileticus. All the conditions were treated as per guidelines.

Imaging methods used were CT and MRI scans. Both scans were used to locate the thrombus; and number, size of any parenchymal lesions.

The etiological work up was done; thrombophilia screening (proteins C and S, anti thrombin III lupus anticoagulant, Anticardiolipin antibodies, factor V Leiden and G20210A mutations when ever feasible).

All treatments systematically recorded and prognosis and outcome was also noted. Prognosis was categorized into two: good (alive) or bad (death).

DATA COLLECTION:

All CT scans and MRIs read by an experienced Radiologist. Patients enrolled after radiological confirmation of CVT. The data regarding laboratory tests and radiological investigations retrieved through medical records of patients. The data regarding neurological examination for stroke severity and disability scores were collected by evaluation of pts during admission & follow up.

FOLLOW-UP:

Follow up visits performed at 3 months, 6 months, at 12 months, preferably by direct interview and observations. If that was not feasible, alternative methods included telephone interview of the patient & sending letters were tried. For patients who were lost to follow-up, the condition on the day of hospital discharge was regarded as the final follow-up. Follow-up data recorded as follows: outcome, recurrent symptomatic sinus thrombosis (new symptoms with new thrombus on repeated venogram or MRI), other thrombotic events, seizure, and headaches requiring bed rest or hospital admissions, severe visual loss (quantified with snellen's chart as <6/60), pregnancy, abortion and current antithrombotic and other treatments.

After collecting the above mentioned data from the clinical records, the data was analyzed for the clinical profile i.e. presence and frequency of various symptoms and signs, incidence of various CT scan abnormalities and involvement of various sinuses and venous systems on MRV/ Angiogram

Patients were classified into poor outcome (death) & good outcome (alive.) These 2 outcomes were compared with each other in terms of age clinical symptoms radiological picture (CT scan) and MRV findings. Patients were also grouped into two subgroups according to site of involvement at MRV i.e. patients with pure sinus involvement, both sinus and venous involvement. The clinical and radiological features were then compared between the subgroups. etiological spectrum of all CVT cases were recorded ,long term

seqelae of pts regarding vision ,SZ, any other residual deficit where available was noted, and we tried to find out recanalization rate of CVT on repeat imaging where ever available.

Statistical analysis

We summarized the demographic data as mean and median. Fisher's exact test when appropriate was performed to analyse the univariate relations between possible prognostic factors and outcome at 12 weeks. As it is likely that different prognostic factors are mutually related, the independent effects of prognostic factors were additionally analyzed with multivariate logistic regression,

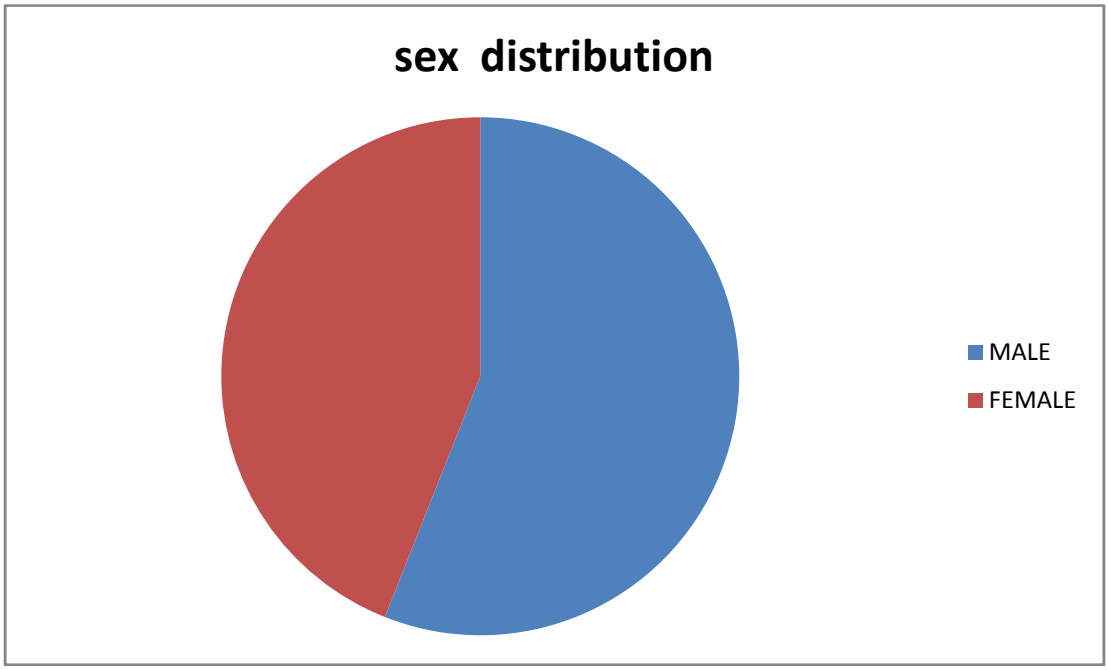
RESULTS

RESULTS

DISTRIBUTION OF PATIENT ACCORDING TO SEX

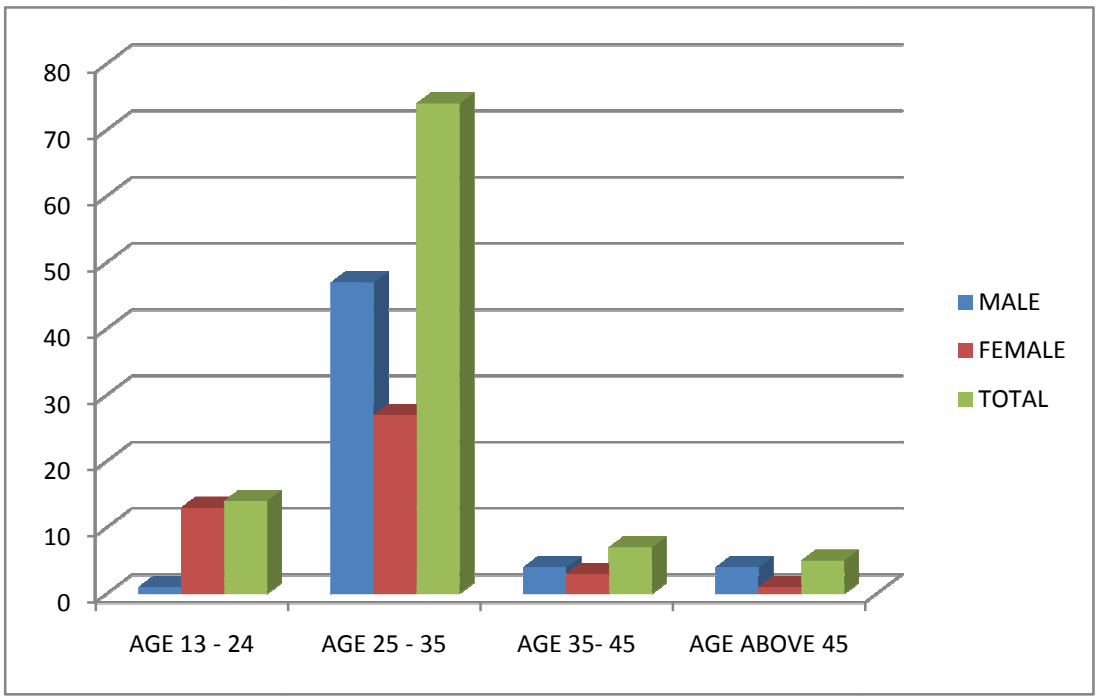
Male: Female ratio: 56:44

Sl. No.	Sex	Total number of cases (n=100)
1	Female	44
2	Male	56



DISTRIBUTION OF PATIENTS ACCORDING TO AGE

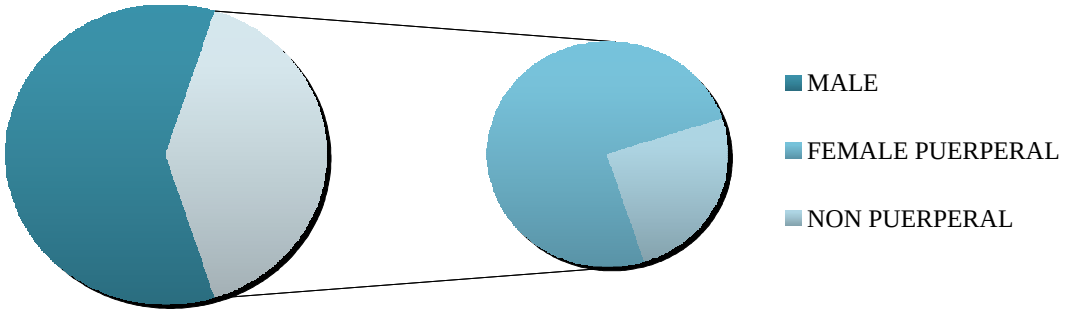
Majority of patients were in second and third decade of their age. 12 patients were in above fourth decade of their age. 4 pts were < 18 years of age. 4 female patients were between 19 and 20 years of age.The mean age was 30.18 with a standard deviation of 13.14. With maximum age 76, minimum age 13 years.



AGE GROUP	MALE	FEMALE	TOTAL CASES
AGE 13 - 24	1	13	14
AGE 24 - 35	47	27	74
AGE 36- 45	4	3	7
AGE ABOVE 45	4	1	5
	56	44	100

TYPES OF CVT IN FEMALE PATIENTS:

FIG 9-SEX INCIDENCE



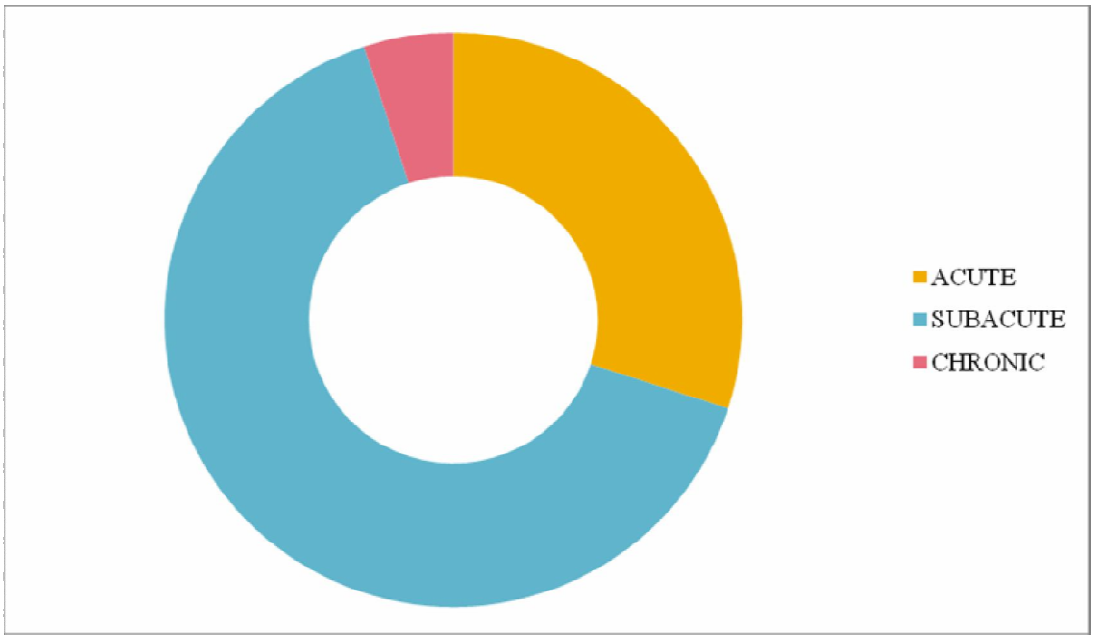
Category	Total number of female cases (n=44)	Percentage in female cases
Puerperal	40	87.5 %
Non puerperal	4	12.5%

There were 56 men (60%) and 44 women (40%) in the study. Out of 44 female patients, 40 (87.5%) were puerperal and 4 (12.5%) were non puerperal CVT. Among 4 non puerperal cases, 1 case was diagnosed as fungal meningitis (mucor mycosis). 1 case was diagnosed as HIV 1 positive. 1 case had TB meningitis.

DISTRIBUTION OF PATIENTS ACCORDING TO DURATION OF SYMPTOMS

Majority of patients (87) had duration of symptoms less than 30 days. A small number of patients (8) had symptom duration of less than 24 hrs. And 5 had symptoms present more than a month

Mode of onset	Percentage
Acute (< 48 hours)	30 %
Sub acute (day 3 – day 30)	65%
Chronic (> 1 month)	5%



ETIOLOGICAL SPECTRUM

Most of the men presenting with CVT had history of binge drinking of alcohol (42 patients) and some dehydration. Most of the female patients got puerperal CVT (40 patients). In non puerperal women infection is the most common cause, with diabetes as risk factor. In 20 patients risk factor could not be identified. Rheumatological evaluation was done as per patient's money availability. Also we found 11 patients had the habit of using smokers. 6 were pan chewers. All these factors contribute to hypercoagulable state.

PCOD, OCP pills use the major risk factors identified which were present in 10 out of 100 cases of each in CVTS. Diarrhea and Ramjan fast season resultant dehydration.

CSOM were identified as a risk factor in 6 cases. Two patients had history of CSF leak as the only etiology. In Procoagulant state serum homocysteine was elevated in 3 cases, protein C and Protein S deficiency in 2 of cases .

ETIOLOGICAL SPECTRUM	PERCENTAGE
Systemic disease	10
Serum homocystine	6
SLE	2
OCP	10
Dehydration	67
Polycythemia	19
Alcohol	51
ANCA	4
Post partum	31
Infective	12
OCP ,Protein S	2
Unknown	20

Infections-12:

In this study of 100 cases of CVT, 12 patients found to have infections both systemic and local.

HIV-2

TBM-2

CSOM-2

Fungal meningitis - 2

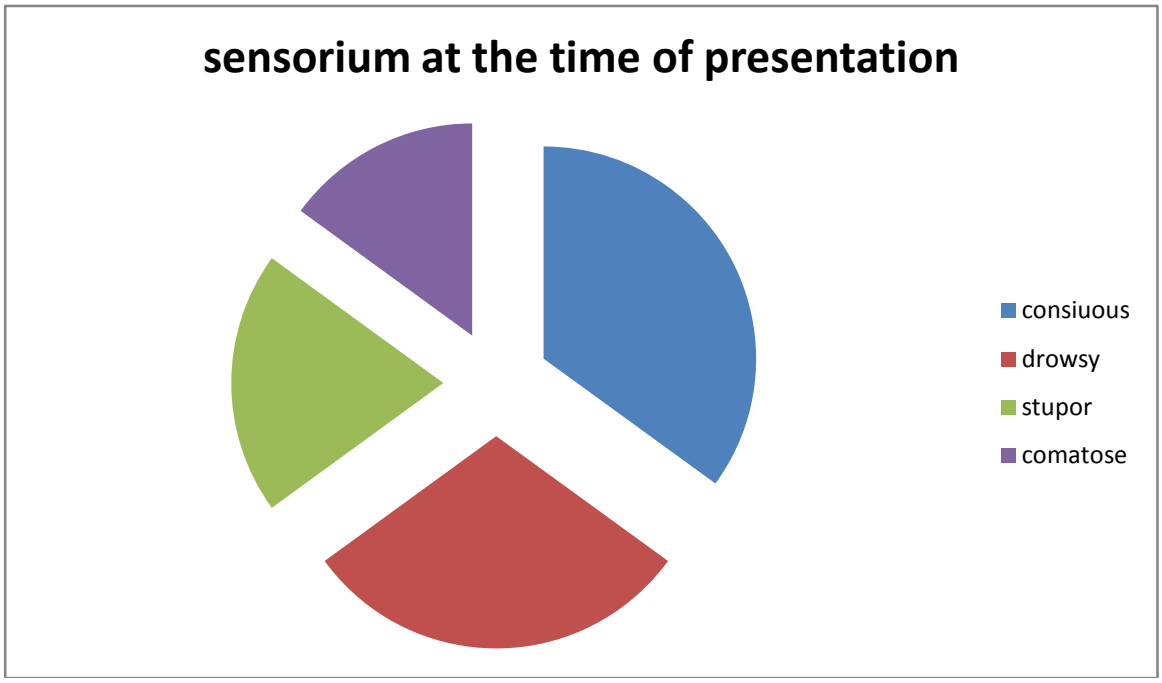
Diarrhoea-4

DISTRIBUTION OF PATIENTS ACCORDING TO SENSORIUM

Majority of patients were in normal sensorium while 30 pts were drowsy. Glasgow Coma Scale (GCS) score was available in all patients. 15 of patients had GCS less than 5 and all of them are immediately intubated or referred from other centers in intubated state.

GLASGOWCOMASCALE

GCS SCORE	No. of patients
< 5	15
< 10	20
10-14	30
15	35
Total	100

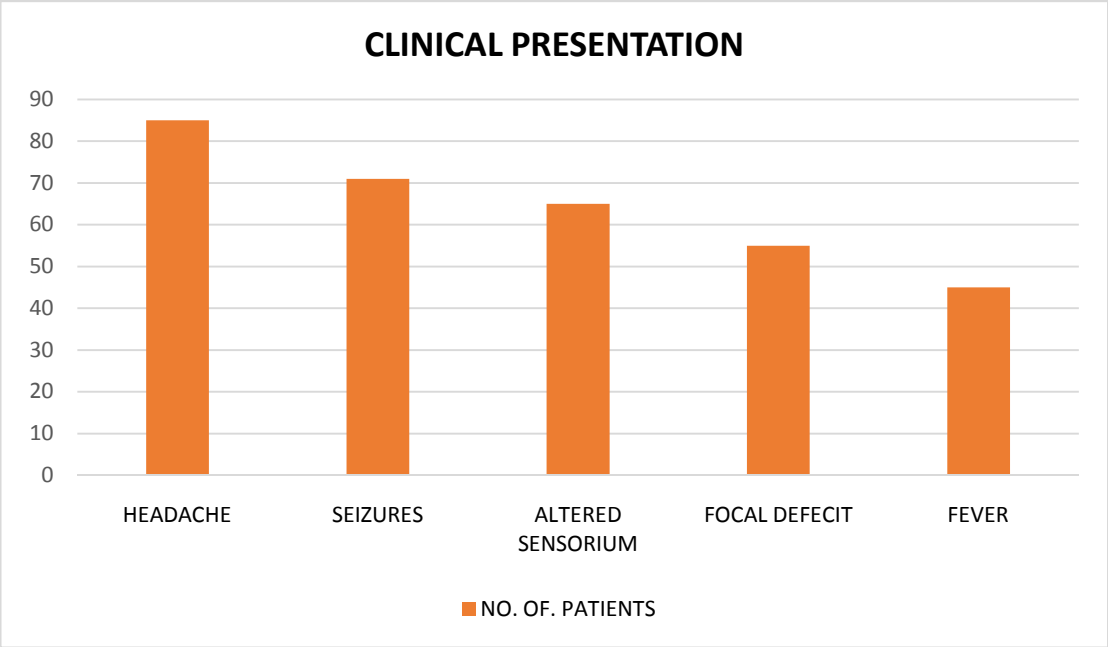


DISTRIBUTION OF PATIENTS ACCORDING TO CLINICAL FEATURES AT PRESENTATION

FOCAL SIGNS	
No focal signs	45
Hemiplegia	36
Hemiplegia with global aphasia	6
Quadriplegia	2
Agraphia ,alexia	2
Cerebellar signs	5
Global aphasia	2
Visual field defects	2
PAPILLOEDEMA	
No	46
Yes	54
SEIZURES	
No seizure	29
Generalized seizures	31
Partial seizures	30
Partial seizures & Status epilepitcus	9
Partial & secondary generalized seizures	10
CRANIAL NERVE PALSY	
No	76
3,4,6,7	2
6	14
6,7	4
UMN FACIAL PALSY	
No	88
Yes	12
HEADACHE	
No	10
yes	83

SYMPTOMS	NO .OF CASES
Headache	83
Convulsions	71
Altered sensorium	65
Focal deficits	55
Fever	45

Out of 100 patients 83 patients had headache at the time of presentation, 55 patients had focal deficits, and 71 patients had seizures and among 12 had generalized seizures, Focal seizures in 30 patients and status epilepticus in 9patients. Facial nerve involvement present in 12 patients. Other cranial palsies present in 18 patients.



INVESTIGATIONS:

HEMOGLOBIN AT THE TIME OF PRESENTATION:

Hb%	Number of cases	Patients alive	Patients dead
2-5	1	1	-
5.1-8	6	5	1
8.1-10	62	60	2
> 10	31	26	5

In my study, maximum number of deaths occurs with HB% > 10 g%, and the percentage of mortality was higher in non anemic group. Higher HB % correlates with the dehydration status, secondary polucythemia. In men , it is mainly due to post binge drinking of alcohol. All patients who died with mild and moderate anaemia were females, and they were in the puerperal group.

CSF ANALYSIS (DONE IN 38 PATIENTS)

CSF changes	Number of Patients
Normal	**16**
Protein rise	**8**
Pleocytosis	**12**
Xanthochromia	**2**

Thiry eight (38) patients were subjected to CSF analysis. CSF analysis done whenever there was suspicion of meningitis. Among 38 patients 16 were normal and abnormality seen in rest 22 patients with pleocytosis being the maximum.

Two patients had TB meningitis with low sugar and high protein content and ADA positive. Two patients have Fungal meningitis with carvenous sinus thrombosis. Two patients had HIV infection. Among them one of them is a known case of HIV and other one is found to be HIV I positive on ELISA.

CT/MRI FINDINGS

Infarction was present in 66 of them out of which 57 had haemorrhagic infarction. 9 patients had non-haemorrhagic infarction. 45 patients had cortical infarction while 5 had deep infarction. One patient had evidence of both cortical and deep infarction. Other than infarction, abnormalities noted on CT scan were mass effect & diffuse edema in among 32pts. 10 pts had cord sign and 7 had empty delta sign.

Lesions in more than one cerebral lobe are more common than single lobe involvement. Temporo occipital lobe lesion was more commonly involved. Among single lobe involvement also temporal lobes were more common. No lesion is seen in 10 CT scans.

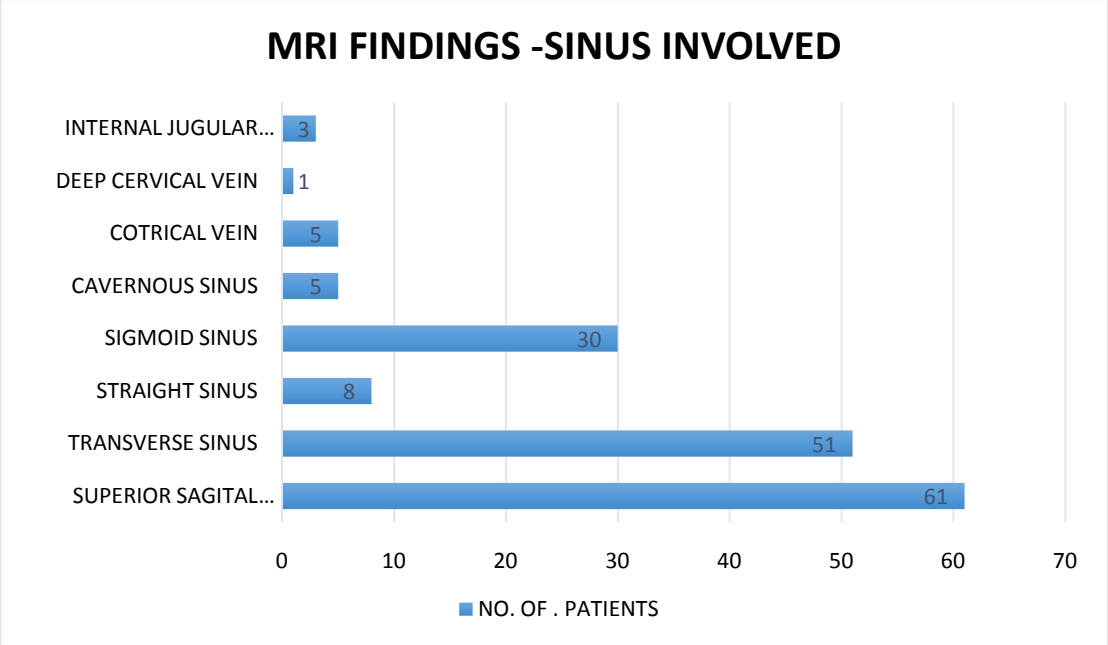
AREA OF INVOLVEMENT	CT FINDINGS
No lesion	10
Frontal lobe lesion	11
Fronto temporal lobe lesion	2
Fronto temporoparietal lobes lesion	2
Frontal & occipital lobes lesion	8
Frontal & parietal lobes lesion	11
Temporal lobe lesion	12
Temporo occipital lobes lesion	18
Temporo ,parieto occipital lobes lesion	2
Temporoparietal lobes lesion	4
Occipital lobe lesion	6
Occipitall & parietal lobes lesion	10
Parietal lobe lesion	2
Diffuse edema	2
Total	100

SIGNS	CT FINDINGS
Cord sign	10
Empty delta sign	7
Dense triangle sign	3
Mass effect	17
Midline shift	17
Bilateral infarct	5
Edema	32
Hemorrhagic infarct	57
Non-haemorrhagic infarct	9
Normal	10

Bilateral hemorrhagic infarct in plain CT

MRV FINDINGS

Superior sagittal sinus (the commonest sinus involved) was involved in 61 patients,(isolated SSS in 7 patients). Total involvement was seen in 11 patients while in other patients anterior, middle and posterior parts involved with various combination of other sinuses. Transverse sinus was the next most common sinus involved 51 patients, (isolated in 4patients) Followed by sigmoid sinus present in 22 pts. Superficial venous system was involved in 5 pts (isolated in 2Pts) while deep venous system was involved in 5pts. Majority (89 pts) of patients had combination of sinuses and veins involvement, 11 pts had only isolated sinus involvement

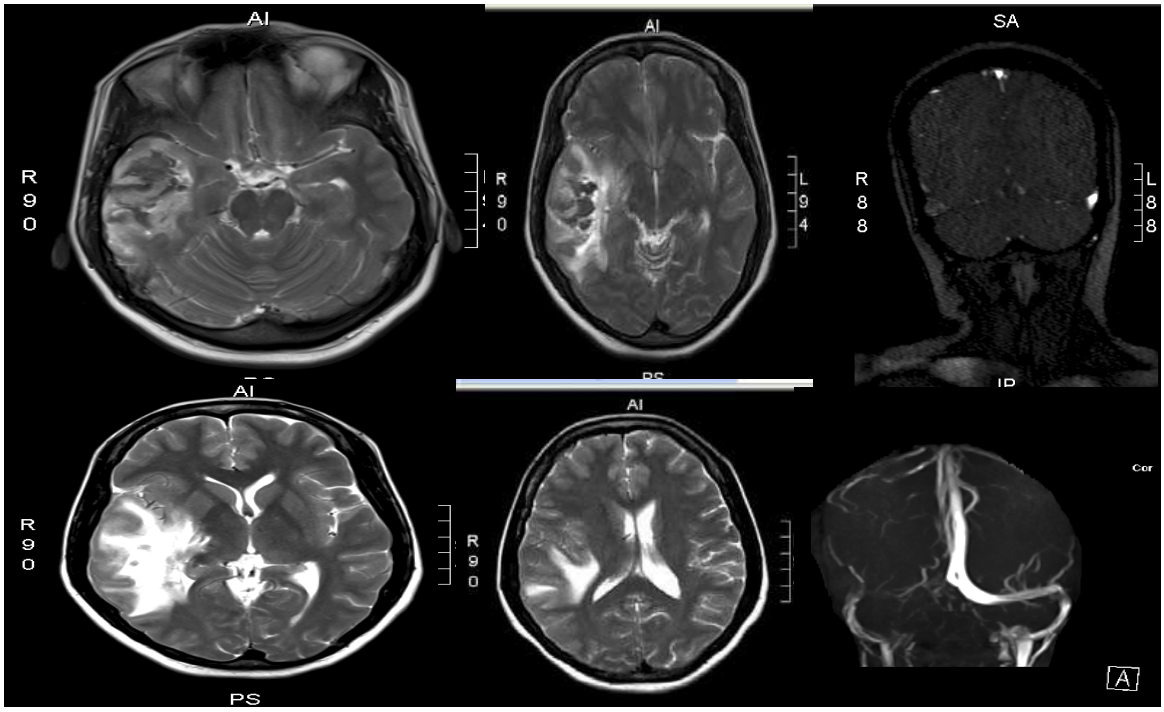


SINUS INVOLVED	NO .OF PATIENTS
Superior sagittal sinus	60
Transverse sinus	50
Straight sinus	8
Sigmoid sinus	30
Cavernous sinus	5
Cortical vein	5
Internal jugular vein	3
Deep cerebral vein	1

COMBINATION OF INVOLVEMENT OF SINUS

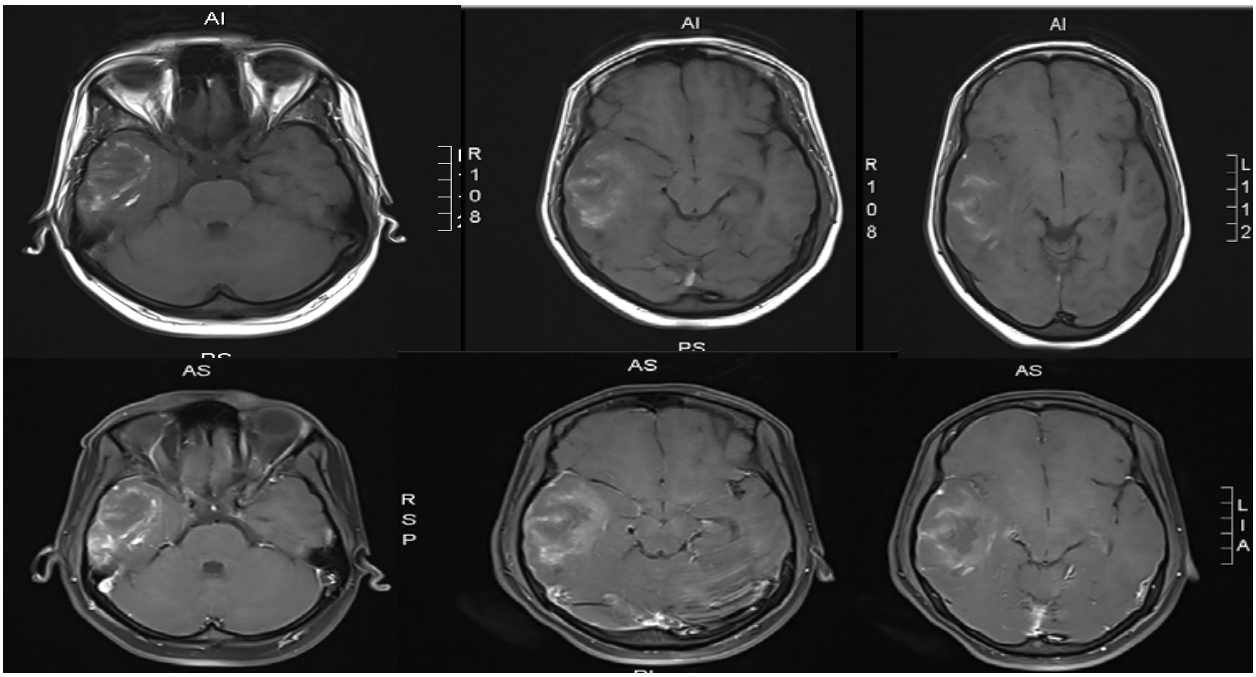
LOCATION OF THROMBUS	PATIENTS
SSS& TS	**14**
SSS,TS,SS(Straight sinus),cortical vein ,IJV	**4**
SSS, TS, SS	**16**
SSS,TS ,Deep veins	**2**
SSS,SS ,Deep veins	**2**
SSS,Cortical veins	**6**
SSS,TS,SS, Deep vein	**4**
SS,IJV,Cavernous sinus	**2**
SSS,TS,SS,Straight sinus	**2**
TS,SS	**10**
TS,SS,Deep vein	**2**
SS,TS, IJV	**6**
TS, SS,IJV	**4**

T2WI



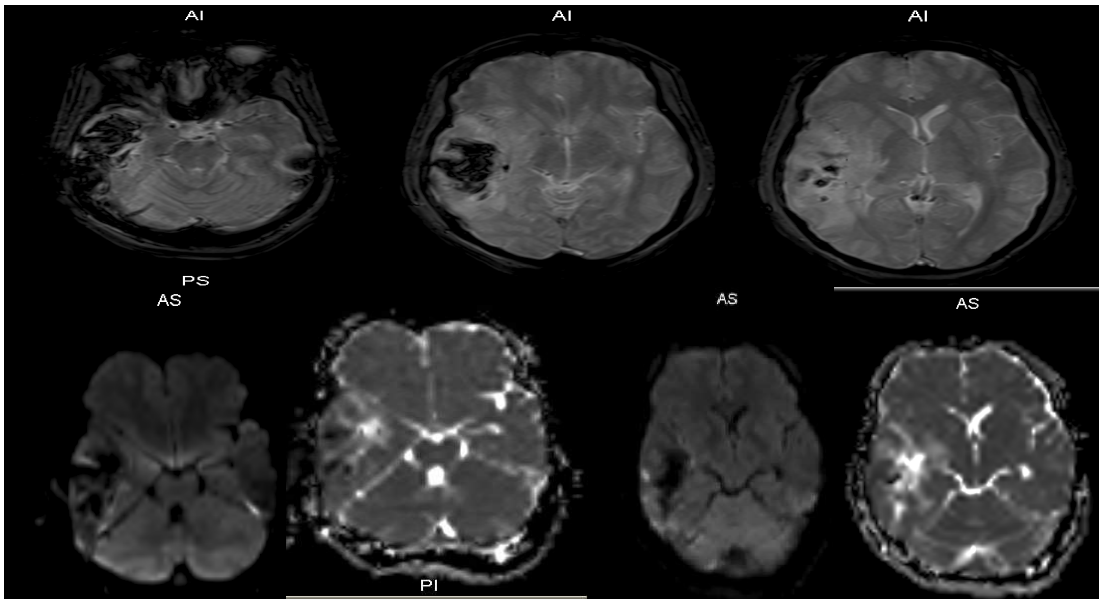
This T2WI showing haemoragic infarct in Right temporal lobe with vasogenic edema , source image showed non filling of contrast in Right sigmoid sinus, MRV showed non filling of Right trswerse sinus

T1WI



T1w & contrast showed haemoragic infarct in Rt temporal lobe with mild constrast enhancement suggestive of subacute stage

SWI



SWI –showing Blooming, ADC, DWI showing both vasogenic, cytotoxic edema

EEG & AED& SZ control

EEG abnormality is present in 20 pts, normal in 56 pts, focal slowing & generalized slowing was the most frequent abnormality, 1 pts had PLEDS, 54 pts had control of SZs with single AED, where as pts 8 required 2 AEDS. And 38 patients don't need AED measures.

EEG	PATIENTS
Normal	56
Focal slowing	2
Generalized slowing	4
G,slowing plus focal spikes	4
Focal spike	4
Generalise spike	4
Pleds	2
Not done	24
Total	100

ANIT EPILEPTIC DRUG	SEIZURE CONTROL
No AED	38
Eptoin	48
Eptoin & valproate	2
Eptoin & levepil	2
Clobazam	2
Clobazam & Eptoin	2
Clobazam & OXC	2
VALPROATE	4
Total	100

TYPE OF TREATMENT RECEIVED

All most all the patients received heparin. 88 of pts received unfractioned heparin, 12 pts received LMWH.IIH type of presentation were received LMWH. 1 pts DAVF underwent embolization. 5 patients underwent decrompression craniotomy. Mechanical ventilator support was needed in 15 patients and 6 patients got good prognosis.

TYPE OF TREATMENT	NO .OF CASES	MORTALITY
ANTI COAGULATION TREATMENT		
IV Heparin	88	5
LMWH	12	4
SURGICAL TREATMENT		
Embolectomy	1	1
Decompression craniotomy done	5	5
MECHANINCAL VENTILATION SUPPORT		
Needed	15	9
Not needed	85	0

CLINICAL AND OTHER POSSIBLE PROGNOSTIC FACTORS RELATED TO OUTCOME AT 12 WEEKS & 6MONTHS AFTER CVT IN 50 PTS

Among 100 patients only 50 pateints were followed up for next 6 months. Among them Female patients were 27 and male were 23. Focal neurological signs were absent in 27 patients and still persist in 23 patients. Headache was still present in 31 patients but intensity was greatly reduced. Seizures was absent in 47 patients and still persist in 3 patients and 1 patient continued AED more than 1 year. Recurrent CT images showed 37 patients still had parenchymal abnormalities at the end of 3rd month.

Prognostic variables	Follow up in 3 month			Follow up in 6th month		
	Number (%)	Male	female	Number (%)	Male	female
Focal neurologic deficit						
Present	23(46%)	10 (20%)	13(26%)	14(28%)	7(14%)	7(14%)
Absent	27 (54%)	13(26%)	14(28%)	36(72%)	16(32%)	20(40%)
Seizures						
Present	4 (8%)	2 (50%)	2 (50%)	2 (50%)	2(80%)	-
Absent	46 (92%)	21(42%)	56%)	48(96%)	21(42%)	27(54%)
Headache						
Present	31(62%)	16 (32%)	15 (30%)	11(22%)	5 (10%)	6 (12%)
Absent	19 (38%)	7 (14%)	12 (24%)	39(89%)	18(32%)	21(42%)
Parenchymal abnormality						
Yes	36(72%)	17(34%)	18(36%)	36(72%)	17(34%)	18(36%)
No	14(28%)	6(12%)	9(18%)	14(28%)	6(12%)	9(18%)

DURATION OF WARFARIN MONTHS

Warfarin duration in most of the cases is upto 6 months 42 patients and 4 of the pts the warfarin was continued >1 year (Procoagulant work up +ve), No intracranial haemorrahage, or any other complication was reported

DURATION OF WARFARIN MONTHS	NO . OF PATIENTS
3	34
6	16
12	4

FOLLOW UP MRV RESULTS:

In the follow up study, almost all the patients were recanlaised at the end of 6 months. Only one patient had persistent thrombus and he was diagnosed as hyperhomocystienemia.

MRV RECANALISATION		
	3 MONTH	6 MONTH
Complete	38 (76%)	44(88%)
Partial	7(14%)	5(10%)
Persistent	5(10%)	1(2%)

Correlation between the site of venous occlusion and clinical parameter

Correlation with etiology showed no constant pattern except that lateral sinus isolated involved in Mastoiditis. Correlation with mode of onset showed no difference in onset whether sinuses alone vs deep venous vs combination of sinuses and veins. no significant difference between presence of various sinuses and venous system the presence and location of infarction .when cortical veins are involved pts were presented with SZs and have intracranial hematoma than when only sinuses were involved.

CLINICAL OUTCOME

Category	No of patients		Mortality
Male	56		2(35.7%)
Female	Puerperal	44	4(9.09%)
	Non-puerperal	4	3(75%)

Overall outcome of the CVT is good. Among 100 patients only 9 deaths occurred. Mostly due to very late referral from other centers because of 'non suspecting' the CVT. Early diagnosis is the key factor for good prognosis in CVT. Early management with heparin is resulted in very good prognosis[1]. All the patients who died had multiple Sinus thrombosis – mostly deep vein like cavernous sinus. And poor GCS at the time of admission is directly related to poor outcome.

Among 2 fungal meningitis patients, none of them recovered. In HIV patient's mortality is 100%. In CVT due to head trauma, 1 death among 2 patients and had 50% mortality.

In puerperal women, 4 deaths among 44 patient and had 9.09% mortality.

DISCUSSION

DISCUSSION

Cerebral venous thrombosis is condition characterized by thrombosis of intracranial veins and sinus which results in parenchymal damage and rise in intracranial pressure. Radiological hallmark of this condition is thrombosis of intracranial sinuses and veins with haemorrhagic infarction and edema with or without evidence of herniation. In this study, total 100 patients with Radilogical features of cerebral venous thrombosis were evaluated. Only 50 patients were followed up over a period of 1year. 56 out of 100 patients were male and remaining was female. This study of 100 patients with CVT cannot give precise information about the real incidence of the disease and cannot make any generalization of the results to whole country. It has been suggested that the incidence of CVT was higher in males. This was not confirmed in the present series, in which Male.Female (56 : 44). This data is not consistent with previous Indian studies viz. Bansal et al (1980)[13], Srinivasan et al [12] (1983), Nagaraja et al [3] (1987). High proportion of post partum CVT patients was also observed by Cantu et al [7] (1996), from Mexico with similar socio-demographic characteristics and economic status of the patients as in India due to referral bias. This finding of high proportion of CVT cases was not replicated in some other studies viz. Deschiens et al [36] (1996) and Daif et al [40] (1995). The possible explanation may be that the etiological factors as well as clinical profile of CVT is in this part of the state different compare to other parts of India More than half of the patients of CVT evaluated were in the second and

third decade of their age . The mean age of the patients was years 30.15 (SD13.14) similar to earlier studies from India (Nagaraja et al [3] 1987) Like all other series, the present one represents a selected group of patients not representative of the numerous causes that have been described.

However, it confirms the fact that the frequency of septic CVT (12/ 100) has markedly declined with the advent of antibiotics. It also confirms the role of oral contraceptives[37] found as the only aetiologic factor in 5 of our patients. This has now led us, as many others to stop oral contraceptives and promptly look for CVT in women presenting with any of the neurological manifestations described in this study, particularly persistent headache, focal deficits or seizures.

In the present study in addition to conventional risk factors Dehydration(65% among male patiets), hyperhomocystenimia(6%), CSF leak (4%), OCP pill use (10%) are significant risk factors , 24% of patients had Anemia , whether this is a reflection of high incidence of anemia in Indian population particularly in pregnant females or anemia is a real risk factor needs further evaluation. In 20/100 cases, no cause could be found, however complete etiological workup could not be completed.

Headache with or without vomiting (82%), remains the main presenting complaint of the patients[3]. The present study was comparable with most other studies like Neki s et al. with 85.5%, Daif et al. with 82% and mehta et al.[74] with 77.8%.

Second most common complaint was seizures (71/100), then altered sensorium (65/100) and Focal deficits (55/100). Papilledema was present in 54 of our cases, was slightly more frequent than in other series: were the major clinical features noted at presentation. Similar findings were noted in the earlier studies [2, 3, 13, 30]. The clinical presentation could be summarized in 3 main patterns, each of them simulating another neurological disease. The most frequent and homogeneous one was the progressive onset of signs of intracranial hypertension corresponding to the "Benign intra-cranial hypertension" or "pseudo-tumor cerebri" syndromes, confirming that sinus thrombosis in 54 % cases these syndrome should not be diagnosed purely on clinical, CSF and CT scan findings without a good quality CT and MRV to rule out the possibility of sinus thrombosis.

Other less common presentations are headache of sudden onset simulating subarachnoid hemorrhage (1 patient). It is therefore clear that CVT has no single clinical presentation and this is why it is necessary to systematically contemplate this diagnosis in order not to overlook it.

Present series most of the patients had good outcome, recanalisation in repeat MRI also achieved.

Recanalization at 3 to 6 months and at 1 year or more

Study, year	No.of Patients	Partial recanal at 3 to 6 mo, no	Complete recanal at 3 to 6 mo, no	Partial recanal at 1 y or more, no.	Complete recanal at 1 y or more, no.
Stolz et al,[10] 2004	37	7	19	7	20
Favrole et al, [11] 2004	28	7	16	NA	NA
Baumgatner et al, [12] 2003	33	15	18	15	18
Strupp et al, [17] 2002	40	NA	NA	12	21
Cakmark et al, [14] 2003	16	12	NA	NA	NA
Present study	**50**	**5**	**44**		

Several reports have emphasized the importance of EEG changes in CVT, the most common pattern being a severe generalised slowing more marked on one side with frequent epileptic activity.[40] in the present series, EEG abnormalities were less severe and they were present in 38 % of cases. Its main interest was to show in a number of patients with focal symptoms a generalised slowing indicating a more diffuse lesion than was clinically suspected. This, however, is in no way specific of CVT. single case showed PLEDS

The present series confirms the fact that isolated single sinus involvement was less common than multiple sinuses involvement, in isolated sinus most frequently involved are SSS and LS Thus in most cases, occlusion involved at least two sinuses or sinus and cerebral veins. Among these, cortical veins were affected slightly more commonly than the deep venous system

These frequent associations probably explain, at least partly, why no good clinico-radiological correlations could be established

Before the introduction of angiography, CVT was diagnosed at autopsy and therefore thought to be most often lethal. In early angiographic series, mortality still ranked between 30% and 50% but in more recent series, it was between 25% and 30% and in our study, it was only 9%. Multiple reasons can explain this decrease, the main one being probably that it is now possible to diagnose "benign" forms of CVT with minimal symptoms and spontaneous recovery. Another reason is that septic thrombosis has, since the use of antibiotics, become both far less frequent and severe. It is also that the introduction of anticoagulant treatment early in the course of the disease has improved the outcome.

Two kinds of sequelae are encountered: blindness /Fieldcut due to optic atrophy/cortical infarcts which should be prevented by early treatment, and focal deficits, usually motor, sometimes associated with epilepsy. Seizures are more frequent when the lesion is anterior to the central sulcus and in patients who have focal deficits. In the VENOPORT study early seizures were associated with sensory deficits and parenchymal lesions on admission CT/MRI. Our study also showed that 80% of the pts had seizure at presentation only 5 patients had long term recurrence , all of them had parenchymal abnormality at presentation. Most of patients presented with seizures were well controlled with single AED

ACUTE ANTITHROMBOTIC TREATMENT: While the overall aim of treatment for CVT is to improve outcome, the immediate goals treatment for CVT are [1,2,3,4]:

●To recanalize the occluded sinus/vein

●To prevent the propagation of the thrombus, namely to the bridging cerebral veins

●To treat the underlying prothrombotic state, in order to prevent venous thrombosis in other parts of the body, particularly pulmonary embolism, and to prevent the recurrence of CVT

The main treatment option to achieve these goals is anticoagulation, using either heparin or low molecular weight heparin (LMWH).

Early anticoagulation: Based upon available published data and guidelines, AHA/ASA guideline recommends the anticoagulation with subcutaneous LMWH or intravenous heparin for adults with symptomatic CVT who have no contraindication. The presence of hemorrhagic venous infarction is not a contraindication for anticoagulant treatment in CVT. 2 studies evidence suggests that subcutaneous LMWH is more effective than unfractionated heparin (UFH), and is at least as safe. Therefore, AHA/ASA suggests subcutaneous LMWH unless the patient is clinically unstable, or invasive interventions such as lumbar puncture or surgery are planned, or there is a contraindication to LMWH, such as renal failure.

Efficacy — although definitive evidence of effectiveness is lacking, there is a general consensus that anticoagulation with UFH or LMWH is

appropriate treatment for acute CVT[2]. As an example, more than 80 percent of the patients in the International Study on Cerebral Vein and Dural Sinus Thrombosis (ISCVT) were treated with anticoagulation [6].

Two randomized controlled trials of anticoagulation in acute CVT have been published

●The Berlin trial of intravenous heparin versus placebo was stopped prematurely because of excess mortality in the placebo arm [7]. Patients randomized to the heparin arm had significantly better outcomes on a not validated composite CVT severity scale than those in the placebo group. The average length from onset of symptoms to anticoagulation treatment, four weeks, was exceptionally long.

●The Dutch trial of subcutaneous nadroparin versus placebo enrolled 60 patients, but excluded those who needed lumbar punctures for the relief of increased intracranial pressure[6]. More patients treated with LMWH followed by oral anticoagulation had a favorable outcome than controls, but the difference between the groups was not statistically significant. Despite randomization, an imbalance at baseline may have favored the placebo group, as there were more cases with isolated intracranial hypertension in the placebo group and more patients with infarcts in the nadroparin group.

UFH	LMWH
Advantages of UFH • Rapidly enters the blood stream and acts swiftly to prevent clot formation • Rapidly wears off when the infusion or injections are stopped • Rapidly reversed by protamine, a UFH antidote, if serious side effects occur • Inexpensive compared with other heparin formulations	**Advantages of LMWH** • Longer and more predicable activity than UFH • Self-administered at home via subcutaneous injection, reducing or eliminating hospital stays • No regular blood monitoring required
Disadvantages of UFH • Frequent blood tests are necessary to ensure correct dosage • IV administration requires hospitalization usually for 5-10 days after blood-clot diagnosis	**Disadvantages of LMWH** • Expensive • Can be uncomfortable to administer, especially if a patient is fearful of needles • Longer activity can complicate reversal, if necessary
Potential Side Effects of UFH • Uncontrolled bleeding (most serious side effect) • Injection site reactions such as redness and irritation • Loss of bone strength • Elevated liver enzymes • Heparin induced thrombocytopenia (HIT)	**Potential Side Effects of LMWH** • Uncontrolled bleeding (most serious side effect) • Injection site reactions such as redness, irritation and bruising • Loss of bone strength (less than UFH) • Elevated liver enzymes • Heparin induced thrombocytopenia (HIT)

In present study also UFH in 87 of pts and LMWH in 12 of pts, based on the idea that an adequate level of anticoagulation is achieved more rapidly with UFH, and then change to LMWH after a few days. UFH is an increased risk of hemorrhagic complications. Other studies have demonstrated that it often takes 24 hours until patients are adequately anticoagulated with UFH, even if a treatment algorithm is used. Thus, the theoretical advantage of more rapid anticoagulation with intravenous UFH is probably rarely realized in practice. The decision to change the type of heparin has been motivated by several reasons. For example, if a patient deteriorates, most notably in the case of a hemorrhagic complication, treating neurologist may decide to switch to another type of heparin. In addition, a switch to LMWH can be made if a patient had improved enough to be mobilized or discharged because LMWH does not require intravenous access.

Factors classically considered of bad prognosis are the rate of evolution of thrombosis,[9] the presence of coma,[10] the age of patients, with a high mortality rate in infancy and in the aged[9] and the involvement of cerebral veins.[41]

Most of the pts who were followed up had re canalized the occluded veins. Only one pt expired in acute phase, Only 1 pt presented with recurrent CVT**[42],** sequelae were both more frequent in patients with focal symptoms than in patients with benign intracranial hypertension. The outcome was otherwise most unpredictable: some acute cases, even with coma, made a remarkably rapid and complete recovery whereas chronic cases often recovered more

slowly and with more frequent sequelae. It is apparent from the study of literature and from the present series that the natural history and prognosis of CVT are highly variable

In this study, attempt was made to correlate the clinical profile with the topographic Radiological substrate like involvement of superficial / deep venous system or the pattern of infarction. There was no significant correlation to evolve a pattern of diagnostic significance, correlating with radiological findings. However predictably, patients with deep venous system involvement and having ganglionic infarction had significantly less incidence of seizures. Patients with involvement of SSS had higher incidence of seizure and lower incidence of headache than those who didn't have SSS involvement. As most of the patients had extensive involvement of cerebral sinovenous system, contribution of degree of involvement of anatomical structures to a particular clinical profile cannot be reliably predicted. For example, high incidence of seizures in patients with SSS involvement may be attributed to the thrombosis from SSS spreading to cerebral veins causing cortical lesions and seizures but when a group of patients with only cerebral venous thrombosis without any sinus thrombosis was analyzed, seizure incidence was not high. Similarly patients with papilloedema did not differ in pathologic Radiological findings when compared to the patient group without papilloedema.

SUMMARY AND CONCLUSIONS

1. Over a period of 1 year, 100 patients of cerebral venous thrombosis were studied. Almost two third (76%) of patients were in 3rd decade of life. This data consistent with most of the earlier Indian studies.
2. Large number of patients 67% in this series had sub acute onset of symptoms i.e. symptom duration (48hours -30 days).
3. Headache is the most common symptom, about 83% of patients had headache. Second most common symptom is new onset seizures 71%, followed by altered sensorium and Focal deficits. Papilledema was present in 54% major clinical features noted.
4. Among male patients, dehydration is the most common cause and most of them had history of binge drinking of alcohol. Second most common cause in men were infection , followed by head trauma
5. Among female patients, puerperium is the most common associated factor and most of them presented with new onset seizures.
6. Cerebral infarction was the most common abnormality noted on CT scan (72%) which was haemorrhagic in 29% of the cases. Deep seated venous infarction (Thalamus and basal ganglionic structure) was seen in 10% of cases
7. On MRV, multiple sinus involvement was the most common presentation. Superior sagittal sinus (the commonest sinus involved) was involved in 61 patients, (isolated SSS in 11 pts).
8. Transverse sinus was the next most common sinus involved 33 pts, (isolated in 4pts) followed by sigmoid sinus 22 pts. Superficial venous system was

involved in 5 patients while deep venous system was involved in 5patients. Majority of patients had combination of sinuses and veins involvement.

9. When attempt was made to correlate the clinical profile with the topographic Radiological substrate like involvement of superficial/deep venous system or the pattern of infarction, there was no significant correlation to evolve a pattern of diagnostic significance, correlating with involvement of sinus
10. CVT is an important and treatable cause of the stroke; risk factors like hyperhomocystenemia**,** OCP use, alcoholism, procoagulant state are increasingly recognized in addition to the conventional risk factors like postpartum state. Procoagulant state and infections are the most common predisposing factors for cerebral venous thrombosis in this study.
11. Prognosis of CVT predominantly determined by early suspicion, diagnosis of CVT and starting early management with anticoagulation treatment.
12. Most of the patients who were followed up had recanalisation of the occluded veins. Complete recanalisation achieved in 88% of the cases. One patient had persistent occlusion and presented with recurrent CVT
13. Imaging plays a key role in diagnosing cerebral venous thrombosis, a condition that can be mimicked by several other neurological entities.
14. The most sensitive diagnostic modality of choice is MRI with MR venography.
15. Multiple risk factors can be present in a single patient

LIMITATIONS OF THE STUDY

LIMITATIONS OF THE STUDY

1. In the study period of 1 year only 50% of cases were followed up for 6 months.
2. Procoagualtion profile workup was so costly and was done only to patients who were affordable.
3. Most male patients had history of binge drinking of alcohol, but not able to find any correlation with the amount of alcohol and development of CVT.
4. Among female patients, about 50% of them got no records of their antenatal period details.
5. Not able to establish the superiority of LMWH against UFH in this study.

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GLASGOW COMA SCALE

Eye response (E)

There are four grades starting with the most severe:

1. No opening of the eye
2. Eye opening in response to pain stimulus. (a peripheral pain stimulus, such as squeezing the lunula area of the patient's fingernail is more effective than a central stimulus such as a trapezius squeeze, due to a grimacing effect).[5]
3. Eye opening to speech. (Not to be confused with the awakening of a sleeping person; such patients receive a score of 4, not 3.)
4. Eyes opening spontaneously

Verbal response (V)

There are five grades starting with the most severe:

1. No verbal response
2. Incomprehensible sounds. (Moaning but no words.)
3. Inappropriate words. (Random or exclamatory articulated speech, but no conversational exchange. Speaks words but no sentences.)
4. Confused. (The patient responds to questions coherently but there is some disorientation and confusion.)
5. Oriented. (Patient responds coherently and appropriately to questions such as the patient’s name and age, where they are and why, the year, month, etc.)

Motor response (M)

There are six grades:

1. No motor response
2. Decerebrate posturing accentuated by pain (extensor response: adduction of arm, internal rotation of shoulder, pronation of forearm and extension at elbow, flexion of wrist and fingers, leg extension, plantarflexion of foot)

3. Decorticate posturing accentuated by pain (flexor response: internal rotation of shoulder, flexion of forearm and wrist with clenched fist, leg extension, plantarflexion of foot)
4. Withdrawal from pain (absence of abnormal posturing; unable to lift hand past chin with supraorbital pain but does pull away when nailbed is pinched)
5. Localizes to pain (purposeful movements towards painful stimuli; e.g., brings hand up beyond chin when supraorbital pressure applied)
6. Obeys commands (the patient does simple things as asked)

Interpretation

Individual elements as well as the sum of the score are important. Hence, the score is expressed in the form "GCS 9 = E2 V4 M3 at 07:35".
Generally, brain injury is classified as:

- Severe, GCS < 8–9
- Moderate, GCS 8 or 9–12 (controversial)[6]
- Minor, GCS ≥ 13.

Tracheal intubation and severe facial/eye swelling or damage makes it impossible to test the verbal and eye responses. In these circumstances, the score is given as 1 with a modifier attached (e.g. "E1c", where "c" = closed, or "V1t" where t = tube). Often the 1 is left out, so the scale reads Ec or Vt. A composite might be "GCS 5tc". This would mean, for example, eyes closed because of swelling = 1, intubated = 1, leaving a motor score of 3 for "abnormal flexion".

PROFORMA

PROFORMA

PROFORMA

NAME: DOA:

AGE: DOD/E:

SEX: male / female Hospital No:

Occupation: Address:

Socio-economic status: Literacy status:

COMPLAINTS:

1. Head ache: yes/no a) Type: frontal/temporal/occipital/diffuse

b) Side: right/left/bilateral

c) Duration:

2. Convulsions: yes/no a) focal/GTCS/focal +GTCS

b) single/status/multiple

c) Duration:

3. Limb weakness: yes/no right/left/both

4. Consciousness: normal/altered

5. Speech disturbances: normal/aphasic

6. Visual disturbances: yes/ no

7. Chronoloogy of symptoms: static/progressive/regressive/fluctuating

8. Neuro psychiatric illness:

9. Non-neurologic symptoms: yes/no

Fever

Vomiting

Diarrhea

Ear ache/ discharge

10. Rheumatological symptoms: yes/no

Arthralgia

Oral ulcers

Skin rashes

Alopecia

Photosensitivity

Others

PAST HISTORY:

DM/SHT/TB/CAD/OCP intake/similar illness/epileptic

PERSONAL HISTORY:

1. Smoker yes/no 2. Alcohol yes/no

3. I.V drug abuse yes/no 4. Pan user yes/no

MENSTRUAL HISTORY: (If female)

Regularity: Married: yes/no Abortions:

IF POSTPARTUM:

a. Duration of pregnancy at delivery Abortion/Pre term/Full term/Post term

b. Place of delivery: Home / Hospital

c. Type of delivery: Normal / Caesarean

d. Antecedent events: Present / Absent

If present, Pre-eclampsia/APH/PPH/Retained placenta

e. Duration between delivery and onset of symptoms < 24 hr/1-10 days/11-20 days/20-30 days/>30 days.

FAMILY HISTORY: DM/ SHT/ stroke/ rheumatologic illness.

CLINICAL FINDINGS:

Pulse BP temp

Pallor: Present / Absent

Icterus: Present / Absent

Cyanosis: Present / Absent

Clubbing: Present / Absent

Pedal edema: Present / Absent

Lymphadenopathy : Present/Absent

Signs of dehydration:

SYSTEMIC EXAMINATION:

Neurological Examination:

1. Glasgow Coma Scale
2. Level of consciousness: Conscious/Drowsy/Stuporus/Coma
3. Speech: Normal/Aphasia
4. Fundi abnormalities: RT LT (Normal/ Papilledema)
5. Gaze paresis
6. Cranial nerve palsies
7. Tone: RT LT (Normal/Increased/ Decreased)

   UL

   LL
8. Power: RT LT (Normal/ Decreased)

   UL

   LL
9. Deep tendon reflexes: RT LT (Normal/Brisk/Sluggish)

   UL

   LL
10. Plantar reflex: RT LT (Flexor/Extensor/Equivocal)
11. Sensory system: RT LT (Normal/Decreased/Cannot be assessed)
12. Cerebellar system: RT LT (Present/Absent/Cannot be tested)

13. Extra pyramidal involvement: Present / Absent

13. Gait abnormalities: Present / Absent

14. Bladder involvement: Present / Absent

15. Signs of meningeal irritation: Present / Absent

CVS: Normal/Abnormal

RS: Normal/Abnormal

P/A: Normal/Abnormal

ENT: 1.Sinusitis - Present / Absent 2.Mastoiditis - Present / Absent

INVESTIGATIONS

Hb: TC: DC: N L M E B ESR: Platelets:

P/S:

BT: CT: PT: INR: APTT:

RBS:

T.Cholesterol:

LDL:

TGL:

Serum urea:

Serum creatinine:

Serum sodium:

Serum potassium:

Serum bilirubin:

ECG:

Urine routine:

Chest X-ray PA view:

CT:

MRV:

ANA (if done):

APLA (if done):

RA factor (if done)

Mechanical ventilation needed : yes or no

TREATMENT DETAILS:

Anti coagulants:

Anti edema measures:

Anti epileptics:

Physiotherapy:

SURGERY DETAILS

De compression craniotomy (if done)

Embolectomy done

OUTCOME:

Improved/ Static/ Deteriorated/Expired.

INFORMATION CHART

INFORMATION SHEET

We are conducting a study on **"A HOSPITAL BASED STUDY ON CLINICAL PROFILE AND OUTCOME OF CEREBRAL VENOUS SINUS THROMBOSIS",** among patients attending Rajiv Gandhi Government General Hospital, Chennai and for that your data will be valuable to us.

The purpose of the study is early diagnosis of CVT in patients with history and physical examination suggestive of CVT.

We are selecting certain cases and if you are found eligible,we me may use your clinical and lab parameters for our study.

The privacy of the patients in the research will be maintained throughout the study. In the event of any publication or presentation resulting from the research, no personal information will be shared.

Taking part in the study is voluntary. You are free to decide whether to participate in this study or to withdraw at any time. Your decision will not result in any loss of benefits to which you are otherwise entitled

The results of this special study may be intimated to you at the end of the study period or during the study if anything is found abnormal which may aid in the management and treatment.

Signature of the investigator Signature of the participant

Date

Place

ஆய்வு தகவல் தாள்

<u>ஆய்வு தலைப்பு</u> : பொது மருத்துவ பிரிவில், மருத்துவ மனையில் உள்ள தகவல்களை கொண்டு தயாரிக்கப்படும் ஆய்வு. மூளை சிரை நாளங்களில் இரத்த உறைவு நோய் உண்டாகும் காரணங்கள் மற்றும் பின் விளைவுகளை கண்டு அறிவதற்கான ஆய்வு.

ஆய்வாளர் பெயர் : மரு. கோ. அரவிந்தன்

ஆய்வு நிலையம் : பொது மருத்துவப்பிரிவு

சென்னை மருத்துவக்கல்லுரி, சென்னை -3

இந்த ஆய்வில் தங்களை பங்கேற்க அழைக்கிறோம். இந்த தகவல் அறிக்கையில் கூறப்பட்டிருக்கும் தகவல்கள் தாங்கள் இந்த ஆராய்ச்சியில் பங்கேற்கலாமா வேண்டாமா என்பதை முடிவு செய்ய உதவியாக இருக்கும். இந்த படிவத்தில் உள்ள தகவல்கள் பற்றி உள்ள சந்தேகங்களை நீங்கள் தயங்காமல் கேட்கலாம்.

இதில் மூளை சிரை நாளங்களில் இரத்த உறைவு நோய் உண்டாகும் காரணங்கள் மற்றும் பின் விளைவுகளை கண்டறியும் ஆராய்ச்சி செய்கிறோம். அதற்கு இரத்த பரிசோதனையும் சீடி மற்றும் எம். ஆர். ஐ ஸ்கேன் அவசியம். அதற்கு தங்களது ஒத்துழைப்புத் தேவை.

நீங்களும் இந்த ஆராய்ச்சியில் பங்கேற்க நாங்கள் விரும்புகிறோம்.முடிவுகளை அல்லது கருத்துகளை வெளியிடும் போதோ அல்லது ஆராய்ச்சியின் போதோ தங்களது பெயரையோ அல்லதுஅடையாளங்களோ வெளியிடமாட்டோம் என்பதையும் தெரிவித்துக் கொள்கிறோம்.

இந்த ஆராய்ச்சியில் பங்கேற்பது தங்களுடைய விருப்பத்தின் பேரில்தான் இருக்கிறது. மேலும் நீங்கள் எந்த நேரமும் இந்த ஆராய்ச்சியில் இருந்து பின் வாங்கலாம் என்பதையும் தெரிவித்துக்கொள்கிறோம்.

இந்த சிறப்புப் பரிசோதனைகளின் முடிவுகளை ஆராய்ச்சியின் போது அல்லது ஆராய்ச்சியின் முடிவில் தங்களுக்கு அறிவிப்போம் என்பதையும் தெரிவித்துக் கொள்கிறோம்.

ஆரய்ச்சியாளர் கையொப்பம் :

தேதி :

பங்கேற்பவரின் கையொப்பம் /

இடது கட்டைவிரல் ரேகை

தேதி :

CONSENT FORM

PATIENT CONSENT FORM

Title	:	HOSPITAL BASED STUDY ON CLINICLA PROFILE OF CEREBRAL VENOUS SINUS THROMBOSIS
Study Centre	:	MADRAS MEDICAL COLLEGE,CHENNAI
Patient's name	:	
Patient's age	:	
Identification number	:	

I confirm that I have understood the purpose of the procedures of the above study. I have had the opportunity to ask questions and all my questions have been answered satisfactorily. □

I understand that my participation in the study is entirely voluntary and that I am free to withdraw from the study at any time without my legal rights being affected □

I understand that sponsors of the study, others working on sponsor's behalf, the ethics committee and the regulatory authorities will not need my permission to look at my health records both in respect of current study and any future research that may be conducted in relation to it; even if I withdraw from the study I agree to this access. However I understand that my identity will not be revealed in any information released to third parties unless required by law. I agree not to restrict the use of any data or results arising from the study. □

I agree to take part in the above the study an to comply with the instructions given during the study and inform about any change in my health status to the investigator. □

I hereby give permission to undergo complete clinical examination and investigations as part of the study. □

Signature of the patient
Patient's name and address

Signature of the investigator
Investigator's name

Place

Date

ஆய்வு ஒப்புதல் படிவம்

<u>ஆய்வு தலைப்பு</u> : பொது மருத்துவ பிரிவில், மருத்துவ மனையில் உள்ள தகவல்களை கொண்டு தயாரிக்கப்படும் ஆய்வு. மூளை சிரை நாளங்களில் இரத்த உறைவு நோய் உண்டாகும் காரணங்கள் மற்றும் பின் விளைவுகளை கண்டு அறிவதற்கான ஆய்வு.

ெயர் : தேதி :

வயது : வெளிநோயாளி எண் :

பால் : ஆராய்ச்சி சேர்க்கை எண் :

இந்த ஆராய்ச்சியின் விவரங்களும் அதன் நோக்கங்களும் முழுமையாக எனக்கு தெளிவாக விளக்கப்பட்டது. எனக்கு விலக்கப்பட்ட விஷயங்களை நான் புரிந்துகொண்டு நான் எனது சம்மதத்தை தெரிவிக்கிறேன்.

இந்த ஆராய்ச்சியில் மூளை சிரை நாளங்களில் இரத்த உறைவு நோய் உண்டாகும் காரணங்கள் மற்றும் பின் விளைவுகளை கண்டு அறிவதற்கான ஆய்வில் பரிசோதனைக்கு மனமார சம்மதிக்கிறேன்.

மேற்கண்ட பரிசோதனையின் போது ஏற்படக்கூடிய பின்விளைவுகளையும் முழுவதும் உணர்ந்து இந்த பரிசோதனைக்கு மனமார சம்மதிக்கிறேன்.

நான் ஆராய்ச்சியாளுடன் ஒத்துழைப்பேன் என்றும், எனக்கு ஏற்படக்கூடிய அசாதாரண நிகழ்வுகள் பற்றியும் உடனடியாக ஆராய்ச்சியாளிடம் தெரிவிப்பேன் என்று உறுதி கூறுகிறேன். இந்த ஆய்விலிருந்து எப்போது வேண்டுமானாலும் எக்காரணமும் கூறாமல் என்னை விடுவித்துக்கொள்ளலாம் என்பதை அறிவேன்.

என்னிடம் இருந்து பெறப்படும் தகவல்களை அரசு, வரைமுறை அதிகாரிகள் ஆகியோர்களுடன் பகிர்ந்துகொள்ள ஆராய்ச்சியாளருக்கு அனுமதி அளிக்கிறேன். என்னுடைய சிகிச்சை கட்டுகளை பார்வையிட உரிமை உண்டு. என்னுடைய தகவல்களின் அடையாளம் ரகசியமாக வைக்கப்படும் என்பதை அறிவேன்.

இந்த ஆராய்ச்சியில் பங்கேற்க தன்னிச்சையமாக முழு மனதுடன் சம்மதிக்கிறேன்.

பங்கேற்பவரின் கையொப்பம் / ரேகை : ஆய்வாளர் கையொப்பம் :

பங்கேற்பவரின் பெயர் : ஆய்வாளர் பெயர் :

இடம் :

தேதி :

URKUND

Urkund Analysis Result

Analysed Document:	thesis.pdf (D42231904)
Submitted:	10/7/2018 6:47:00 PM
Submitted By:	aravind2good@gmail.com
Significance:	6 %

Sources included in the report:

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Instances where selected sources appear:

28

PLAGIARISM CERTIFICATE

This is to certify that this dissertation work titled **“A HOSPITAL BASED STUDY ON CLINICAL PROFILE AND OUTCOME OF CEREBRAL VENOUS SINUS THROMBOSIS”** of the candidate **Dr.G.ARAVINDAN** with registration Number 201611002 for the award of M.D in the branch of **GENERAL MEDICINE.** I personally verified the urkund.com website for the purpose of plagiarism check. I found that the uploaded thesis file contains from introduction to conclusion pages and result shows 6 percentage of plagiarism in the dissertation.

Guide & Supervisor sign with Seal.

ETHICAL COMMITTEE

APPROVAL FORM

INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE, CHENNAI 600 003

EC Reg.No.ECR/270/Inst./TN/2013
Telephone No.044 25305301
Fax: 011 25363970

CERTIFICATE OF APPROVAL

To
Dr.G.Aravindan
Post Graduate in M.D. General Medicine
Institute of Internal Medicine
Madras Medical College
Chennai 600 003

Dear Dr.G.Aravindan,

The Institutional Ethics Committee has considered your request and approved your study titled **"A HOSPITAL BASED STUDY ON CLINICAL PROFILE AND OUTCOME OF CEREBRAL VENOUS SINUS THROMBOSIS " - NO.10082017**

The following members of Ethics Committee were present in the meeting hold on **01.08.2017** conducted at Madras Medical College, Chennai 3

1. Prof.Dr.C.Rajendran, MD., :Chairperson
2. Prof.R.Narayana Babu,MD.,DCH.,Dean, MMC,Ch-3 : Deputy Chairperson
3. Prof.Sudha Seshayyan,MD., Vice Principal,MMC,Ch-3: Member Secretary
4. Prof.N.Gopalakrishnan,MD,Director,Inst.of Nephrology,MMC,Ch : Member
5. Prof.S.Mayilvahanan,MD,Director,Inst. of Int.Med,MMC, Ch-3 : Member
6. Prof.Rema Chandramohan,Prof.of Paediatrics,ICH,Chennai : Member
7. Prof. Susila, Director, Inst. of Pharmacology,MMC,Ch-3: Member
8. Prof.K.Ramadevi,MD., Director, Inst. of Bio-Chemistry,MMC,Ch-3 : Member
9. Thiru S.Govindasamy, BA.,BL,High Court,Chennai : Lawyer
10. Tmt.Arnold Saulina, MA.,MSW., :Social Scientist
11. Tmt.J.Rajalakshmi, JAO,MMC, Ch-3 : Lay Person

We approve the proposal to be conducted in its presented form.

The Institutional Ethics Committee expects to be informed about the progress of the study and SAE occurring in the course of the study, any changes in the protocol and patients information/informed consent and asks to be provided a copy of the final report.

Member Secretary – Ethics Committee

MEMBER SECRETARY
INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE
CHENNAI-600 003

MASTER CHART

S.NO	AGE	SEX	ONSET	P / NP	COMPLAINTS							COMORBID STATU	DRUG ABUSE	SIGNS								OUTCOME
					HEADACHE	FOCAL DEFICIT	SEIZURE	CONSCIOUSNE	FEVER	EARDISCHAGE	DIARROHEA			PALLOR	VITALS	HEMIPLEGIA	CRANIALNERV	NECKSTIFFNE		FUNDUS	OTHERSYSTEM	
1	22	F	SA	P	+	-	-	D	+	-	-	PIH	-	+++	S	-	9,10	+		PE	-	A
2	29	M	SA	NP	+	+	FO/GT	CO	-	-	-	INS	S/A	-	US	LH	-	+		PE	-	D
3	25	F	SA	P	+	-	GT	S	+	-	-	SEI	-	+	S	-	-	-		-	-	A
4	21	M	A	NP	+	+	GT	C	-	-	-	-	A	-	S	LH	-	-		DB	-	A
5	31	M	SA	NP	+	+	GT	S	+	-	-	HT	S/A	-	US	RH	6	+		PE	-	A
6	28	M	CH	NP	+	-	-	C	+	-	-	HIV	PAN	-	S	-		-		N	-	D
7	34	M	SA	NP	+	+	FO/GT	D	-	-	-	-	S/A	-	S	LH	-	+		DB	11	A
8	55	F	A	NP	-	+	-	C	-	-	+	HT/DM	-	+	S	RH	-	-		-	-	A
9	40	M	SA	NP	+	+	GT	D	-	-	-	-	S/A	-	S	LH	3	-		-	-	A
10	27	M	A	NP	+	-	-	C	+	-	-	TB	S/A	-	S	-	6	+		PE	RALES	A
11	32	M	SA	NP	-	+	FO/GT	S	+	+	-	CSOM	-	++	S	RH	8	-		-	CSOM	A
12	20	F	A	P	+	-	GT	S	-	-	-	-	-	+	S	-	6	+		PE	-	A
13	28	M	SA	NP	+	+	SE	CO	-	-	-	BA	S/A	-	US	FB	6	+		PE	WHEEZ	D
14	42	M	SA	NP	+	+	GT	D	-	-	-	HT	S	-	S	LH	-	-		-	-	A
15	40	M	A	NP	+	+	FO/GT	D	+	-	-	-	S/A	-	US	RH	6	+		PE	-	D
16	36	M	SA	NP	+	-	GT	S	+	-	-	-	-	+	US	-	-	+		DB	-	A

17	23	F	SA	P	+	-	SE	CO	-	-	-	-	-	++	US	-	7	-		-	-	D
18	26	F	A	P	+	-	GT	D	-	-	-	-	-	++	S	-	-	-		-	-	A
19	31	M	CH	NP	+	+	FO	C	-	-	-	APLA	PAN	+		RH	-	-		DB	-	A
20	25	F	SA	P	+	-	FO	C	+	-	-	RHD	-	+		-	-	-		DB	MDM	A

					COMPLAINTS									SIGNS								OUTCOME
	AGE	SEX	ONSET	P / NP	HEADACHE	FOCAL DEFICIT	SEIZURE	CONSCIOUSNESS	FEVER	EARDISCHAGE	DIARROHEA	COMORBIDSTATU	DRUGABUSE	PALLOR	VITALS	HEMIPLEGIA	CRANIALNERV	NECKSTIFFNES		FUNDUS	OTHERSYSTEM	
21	20	M	SA	NP	-	-	-	C	-	-	-	-	PAN	-		-	-	-		-	-	A
22	22	F	SA	P	+	+	GT	D	+	-	-	-	-	++		RH	-	+		DB	-	A
23	21	F	A	P	+	-	FO/GT	S	+	-	-	-	-	++		-	-	-		DB	-	A
24	27	M	SA	NP	+	+	GT	D	-	-	-	TB	S	-		LH	7,9,10	-		-	RALES	A
25	32	M	SA	NP		+	FO	C	-	-	-	-	S/A	-		LH	-	-		-	-	A
26	43	F	CH	NP	-	-	SE	S	+	-	-	DM	-	-		-	6	+		PE	-	A
27	27	F	SA	P	+	+	GT	D	+	-	-	-	PAN	-		LH	7	-		-	-	A
28	26	M	A	NP	+	+	-	C	-	-	-	-	-	-		RH	-	-		-	-	A
29	33	M	SA	NP	+	+	GT	CO	+	-	-	HIV	PAN	-		RH	-	+		DB	-	D
30	25	F	A	P	+	-	-	C	-	-	-	-	-	+		-	-	-		-	-	A
31	30	M	SA	NP	+	+	FO	C	-	-	-	-	S	-		LH	-	-		DB	-	A
32	24	F	A	P	+	-	FO/GT	CO	-	-	-	PIH	-	+		-	-	-		-	-	D

33	38	M	SA	NP	+	+	GT	D	-	-	-	-	S/A	-		FB	-	-		-	-	A
34	41	M	SA	NP	+	+	FO/GT	D	+	-	-	HT	S	-		RH	3	+		DB	-	A
35	36	M	A	NP	+	+	GT	D	+	-	-	BA	S/A	-		LH	-	-		-	WHEEZ	A
36	26	F	SA	P	+	-	FO	C	-	-	-	-	-	++		-	-	-		-	-	A
37	29	F	SA	P	+	-	GT	CO	+	-	-	-	-	+		-	-	-		DB	-	D
38	22	F	A	P	+	-	-	C	-	-	-	-	-	+		-	-	-		-	-	A
39	36	M	SA	NP	+	+	FO/GT	S	+	-	-	CSOM	PAN	-		LH	8	+		DB	CSOM	A
40	29	M	SA	NP	+	-	FO	C	-	-	-	-	S/A	-		-	-	-		-	-	A
41	22	F	SA	P	+	-	-	D	+	-	-	PIH	-	+++	S	-	9,10	+		PE	-	A
42	29	M	SA	NP	+	+	FO/GT	CO	-	-	-	INS	S/A	-	US	LH	-	+		PE	-	D
43	25	F	SA	P	+	-	GT	S	+	-	-	SEI	-	+	S	-	-	-		-	-	A
44	21	M	A	NP	+	+	GT	C	-	-	-	-	A	-	S	LH	-	-		DB	-	A
45	31	M	SA	NP	+	+	GT	S	+	-	-	HT	S/A	-	US	RH	6	+		PE	-	A
46	28	M	CH	NP	+	-	-	C	+	-	-	HIV	PAN	-	S	-		-		N	-	D
47	34	M	SA	NP	+	+	FO/GT	D	-	-	-	-	S/A	-	S	LH	-	+		DB	11	A
48	55	F	A	NP	-	+	-	C	-	-	+	HT/DM	-	+	S	RH	-	-		-	-	A
49	40	M	SA	NP	+	+	GT	D	-	-	-	-	S/A	-	S	LH	3	-		-	-	A
50	27	M	A	NP	+	-	-	C	+	-	-	TB	S/A	-	S	-	6	+		PE	RALES	A
51	32	M	SA	NP	-	+	FO/GT	S	+	+	-	CSOM	-	++	S	RH	8	-		-	CSOM	A
52	20	F	A	P	+	-	GT	S	-	-	-	-	-	+	S	-	6	+		PE	-	A
53	28	M	SA	NP	+	+	SE	CO	-	-	-	BA	S/A	-	US	FB	6	+		PE	WHEEZ	D

54	42	M	SA	NP	+	+	GT	D	-	-	-	HT	S	-	S	LH	-	-		-	-	A
55	40	M	A	NP	+	+	FO/GT	D	+	-	-	-	S/A	-	US	RH	6	+		PE	-	D
56	36	M	SA	NP	+	-	GT	S	+	-	-	-	-	+	US	-	-	+		DB	-	A
57	23	F	SA	P	+	-	SE	CO	-	-	-	-	-	++	US	-	7	-		-	-	D
58	26	F	A	P	+	-	GT	D	-	-	-	-	-	++	S	-	-	-		-	-	A
59	31	M	CH	NP	+	+	FO	C	-	-	-	APLA	PAN	+		RH	-	-		DB	-	A
60	25	F	SA	P	+	-	FO	C	+	-	-	RHD	-	+		-	-	-		DB	MDM	A
61	20	M	SA	NP	-	-	-	C	-	-	-	-	PAN	-		-	-	-		-	-	A
62	22	F	SA	P	+	+	GT	D	+	-	-	-	-	++		RH	-	+		DB	-	A
63	21	F	A	P	+	-	FO/GT	S	+	-	-	-	-	++		-	-	-		DB	-	A
64	27	M	SA	NP	+	+	GT	D	-	-	-	TB	S	-		LH	7,9,10	-		-	RALES	A
65	32	M	SA	NP		+	FO	C	-	-	-	-	S/A	-		LH	-	-		-	-	A
66	43	F	CH	NP	-	-	SE	S	+	-	-	DM	-	-		-	6	+		PE	-	A
67	27	F	SA	P	+	+	GT	D	+	-	-	-	PAN	-		LH	7	-		-	-	A
68	26	M	A	NP	+	+	-	C	-	-	-	-	-	-		RH	-	-		-	-	A
69	33	M	SA	NP	+	+	GT	CO	+	-	-	HIV	PAN	-		RH	-	+		DB	-	D
70	25	F	A	P	+	-	-	C	-	-	-	-	-	+		-	-	-		-	-	A
71	30	M	SA	NP	+	+	FO	C	-	-	-	-	S	-		LH	-	-		DB	-	A
72	24	F	A	P	+	-	FO/GT	CO	-	-	-	PIH	-	+		-	-	-		-	-	D
73	38	M	SA	NP	+	+	GT	D	-	-	-	-	S/A	-		FB	-	-		-	-	A
74	41	M	SA	NP	+	+	FO/GT	D	+	-	-	HT	S	-		RH	3	+		DB	-	A

75	36	M	A	NP	+	+	GT	D	+	-	-	BA	S/A	-		LH	-	-		-	WHEEZ	A
76	26	F	SA	P	+	-	FO	C	-	-	-	-	-	++		-	-	-		-	-	A
77	29	F	SA	P	+	-	GT	CO	+	-	-	-	-	+		-	-	-		DB	-	D
78	22	F	A	P	+	-	-	C	-	-	-	-	-	+		-	-	-		-	-	A
79	36	M	SA	NP	+	+	FO/GT	S	+	-	-	CSOM	PAN	-		LH	8	+		DB	CSOM	A
80	29	M	SA	NP	+	-	FO	C	-	-	-	-	S/A	-		-	-	-		-	-	A
81	22	F	SA	P	+	-	-	D	+	-	-	PIH	-	+++	S	-	9,10	+		PE	-	A
82	29	M	SA	NP	+	+	FO/GT	CO	-	-	-	INS	S/A	-	US	LH	-	+		PE	-	D
83	25	F	SA	P	+	-	GT	S	+	-	-	SEI	-	+	S	-	-	-		-	-	A
84	21	M	A	NP	+	+	GT	C	-	-	-	-	A	-	S	LH	-	-		DB	-	A
85	31	M	SA	NP	+	+	GT	S	+	-	-	HT	S/A	-	US	RH	6	+		PE	-	A
86	28	M	CH	NP	+	-	-	C	+	-	-	HIV	PAN	-	S	-		-		N	-	D
87	34	M	SA	NP	+	+	FO/GT	D	-	-	-	-	S/A	-	S	LH	-	+		DB	11	A
88	55	F	A	NP	-	+	-	C	-	-	+	HT/DM	-	+	S	RH	-	-		-	-	A
89	40	M	SA	NP	+	+	GT	D	-	-	-	-	S/A	-	S	LH	3	-		-	-	A
90	27	M	A	NP	+	-	-	C	+	-	-	TB	S/A	-	S	-	6	+		PE	RALES	A
91	32	M	SA	NP	-	+	FO/GT	S	+	+	-	CSOM	-	++	S	RH	8	-		-	CSOM	A
92	20	F	A	P	+	-	GT	S	-	-	-	-	-	+	S	-	6	+		PE	-	A
93	28	M	SA	NP	+	+	SE	CO	-	-	-	BA	S/A	-	US	FB	6	+		PE	WHEEZ	D
94	42	M	SA	NP	+	+	GT	D	-	-	-	HT	S	-	S	LH	-	-		-	-	A
95	40	M	A	NP	+	+	FO/GT	D	+	-	-	-	S/A	-	US	RH	6	+		PE	-	D

96	36	M	SA	NP	+	-	GT	S	+	-	-	-	-	+	US	-	-	+		DB	-	A
97	23	F	SA	P	+	-	SE	CO	-	-	-	-	-	++	US	-	7	-		-	-	D
98	26	F	A	P	+	-	GT	D	-	-	-	-	-	++	S	-	-	-		-	-	A
99	31	M	CH	NP	+	+	FO	C	-	-	-	APLA	PAN	+		RH	-	-		DB	-	A
100	25	F	SA	P	+	-	FO	C	+	-	-	RHD	-	+		-	-	-		DB	MDM	A

Investigations

S.NO	SUGAR	UREA	CREATININE	LFT	HB%	PLATELETS	CSF	CT SCAN		MRI+MRV FINDINGS									PT/INR	HOMOCYSTEIN	OTHERS	OUTCOME
								DIRECT SIGNS	INDIRECT SIGNS	SSS	TS	STS	SS	CST	CV	IJV	DCV	MASTOIDITI				
1	94	84	2.1	N	4	2.2	N	N	NHI	+	+		+			+		+	1.37	N	N	A
2	110	24	0.7	N	12	3.2	-	EDS	HI	+	+	+							0.9	HI	N	D
3	100	32	0.8	N	8.2	3.1	P	N	N	+									0.92	N	ACL	A
4	98	35	0.9	N	17.2	2.6	-	CS	E	+	+								1.13	N	N	A
5	112	33	0.8	N	14	3.5	N		HI	+									1.21	N	-	A
6	84	34	0.8	N	12	3.1	N	DTS	BHI	+									1.12	HI	-	D
7	111	28	0.7	N	13	2.3	-		HI		+								1.27	HI	-	A
8	234	110	3.2	N	9	2.4	-		NHI		+								1.27	N	-	A
9	96	42	1.1	N	12	2.6	-		MS/ME	+	+		+	+					1.15	HI	-	A
10	99	32	1	N	13	2.8	p/c/x	EDS	HI		+								1.18	N	-	A

11	103	28	0.8	N	7.8	2.4	C		HI/E	+	+				+				1.15	HI	-	A
12	102	26	0.7	N	8	2.8	-		HI	+									1.13	N	-	A
13	121	82	1.8	N	15	3.6	-		ME/E		+								1.21	HI	-	D
14	109	30	0.8	N	13	3.1	-		HI	+									1.12	N	-	A
15	106	28	0.7	N	14	2.6	C		MS/E		+								1.27	HI	-	D
16	98	26	0.7	N	10	3.4	N		HI	+									1.27	N	N	A
17	120	32	0.9	N	7.2	2.9	-		ME/NS	+									1.37	N	N	D
18	131	36	1	N	7.6	3.4	-	N	N		+								0.9	N	-	A
19	85	40	1.2	N	8.4	2.4	-		HI	+									1.22	N	ACL	A
20	76	44	1.1	N	9.6	3.1	N		HI										1.11	N	N	A

S.NO	SUGAR	UREA	CREATININE	LFT	HB%	PLATELETS	CSF	CT SCAN		MRI+MRV FINDINGS									PT/INR	HOMOCYSTEIN	OTHERS	OUTCOME
								DIRECT SIGNS	INDIRECT SIGNS	SSS	TS	STS	SS	CST	CV	IJV	DCV	MASTOIDITI				
21	75	38	1	N	14	3.8	-		HI		+								1.22	HI		A
22	96	42	1.1	N	7.2	3.5	N	CS	HI	+									1.1	N	-	A
23	113	36	0.9	N	7.1	3.6	-		BHI/E	+	+								1.3	N	-	A
24	112	36	0.8	N	12	2.6	P/C/X		HI			+							0.9	HI	-	A
25	86	28	0.7	N	14	3.8	-	CS	HI	+									1.25	HI	-	A
26	116	44	1.1	N	13	3.1	C		HI		+								1.1	N	-	A
27	110	38	0.9	N	12	2.9	C		HI	+									1.21	N	-	A

28	87	36	0.8	N	13	3.4	-		HI		+								1.1	N	-	A
29	99	48	1.2	N	13	1.9	N		ME/E	+									0.9	HI	-	D
30	82	42	1.1	N	9.4	1.9	-	EDS	HI	+									0.9	N	N	A
31	93	40	1	N	13	3.2	-		HI		+								1.13	N	-	A
32	81	96	2.8	N	9.8	2.3	-		MS/E	+	+								1.21	HI	-	D
33	79	26	0.7	N	14	2.7	-		NHI		+	+							1.12	HI	-	A
34	115	46	1.2	N	13	2.4	P	N	N					+					1.27	HI	-	A
35	114	36	0.8	N	12	2.9	P		HI	+									1.27	N	-	A
36	73	28	0.7	N	7.8	2.6	-		HI		+								1.15	N	N	A
37	74	40	0.9	N	9.2	3.5	N	CS	ME/MS	+									1.18	HI	-	D
38	82	72	1.7	N	9.4	2.6	-		HI	+									1.15	N	-	A
39	118	28	0.7	N	12	3.1	P		BHI/E		+				+			+	1.1	HI	C/S	A
40	86	36	0.9	N	14	3.4	-		HI	+									0.9	HI	-	A
41	75	38	1	N	14	3.8	-		HI		+								1.22	HI		A
42	96	42	1.1	N	7.2	3.5	N	CS	HI	+									1.1	N	-	A
43	113	36	0.9	N	7.1	3.6	-		BHI/E	+	+								1.3	N	-	A
44	112	36	0.8	N	12	2.6	P/C/X		HI			+							0.9	HI	-	A
45	86	28	0.7	N	14	3.8	-	CS	HI	+									1.25	HI	-	A
46	116	44	1.1	N	13	3.1	C		HI		+								1.1	N	-	A
47	110	38	0.9	N	12	2.9	C		HI	+									1.21	N	-	A
48	87	36	0.8	N	13	3.4	-		HI		+								1.1	N	-	A
49	99	48	1.2	N	13	1.9	N		ME/E	+									0.9	HI	-	D

50	82	42	1.1	N	9.4	1.9	-	EDS	HI	+									0.9	N	N	A
51	93	40	1	N	13	3.2	-		HI		+								1.13	N	-	A
52	81	96	2.8	N	9.8	2.3	-		MS/E	+	+								1.21	HI	-	D
53	79	26	0.7	N	14	2.7	-		NHI		+	+							1.12	HI	-	A
54	115	46	1.2	N	13	2.4	P	N	N					+					1.27	HI	-	A
55	114	36	0.8	N	12	2.9	P		HI	+									1.27	N	-	A
56	73	28	0.7	N	7.8	2.6	-		HI		+								1.15	N	N	A
57	74	40	0.9	N	9.2	3.5	N	CS	ME/MS	+									1.18	HI	-	D
58	82	72	1.7	N	9.4	2.6	-		HI	+									1.15	N	-	A
59	118	28	0.7	N	12	3.1	P		BHI/E		+				+			+	1.1	HI	C/S	A
60	86	36	0.9	N	14	3.4	-		HI	+									0.9	HI	-	A
61	94	84	2.1	N	4	2.2	N	N	NHI	+	+		+			+		+	1.37	N	N	A
62	110	24	0.7	N	12	3.2	-	EDS	HI	+	+	+							0.9	HI	N	D
63	100	32	0.8	N	8.2	3.1	P	N	N	+									0.92	N	ACL	A
64	98	35	0.9	N	17.2	2.6	-	CS	E	+	+								1.13	N	N	A
65	112	33	0.8	N	14	3.5	N		HI	+									1.21	N	-	A
66	84	34	0.8	N	12	3.1	N	DTS	BHI	+									1.12	HI	-	D
67	111	28	0.7	N	13	2.3	-		HI		+								1.27	HI	-	A
68	234	110	3.2	N	9	2.4	-		NHI		+								1.27	N	-	A
69	96	42	1.1	N	12	2.6	-		MS/ME	+	+		+	+					1.15	HI	-	A
70	99	32	1	N	13	2.8	p/c/x	EDS	HI		+								1.18	N	-	A
71	103	28	0.8	N	7.8	2.4	C		HI/E	+	+				+				1.15	HI	-	A

72	102	26	0.7	N	8	2.8	-		HI	+									1.13	N	-	A
73	121	82	1.8	N	15	3.6	-		ME/E		+								1.21	HI	-	D
74	109	30	0.8	N	13	3.1	-		HI	+									1.12	N	-	A
75	106	28	0.7	N	14	2.6	C		MS/E		+								1.27	HI	-	D
76	98	26	0.7	N	10	3.4	N		HI	+									1.27	N	N	A
77	120	32	0.9	N	7.2	2.9	-		ME/NS	+									1.37	N	N	D
78	131	36	1	N	7.6	3.4	-	N	N		+								0.9	N	-	A
79	85	40	1.2	N	8.4	2.4	-		HI	+									1.22	N	ACL	A
80	76	44	1.1	N	9.6	3.1	N		HI										1.11	N	N	A
81	94	84	2.1	N	4	2.2	N	N	NHI	+	+		+			+		+	1.37	N	N	A
82	110	24	0.7	N	12	3.2	-	EDS	HI	+	+	+							0.9	HI	N	D
83	100	32	0.8	N	8.2	3.1	P	N	N	+									0.92	N	ACL	A
84	98	35	0.9	N	17.2	2.6	-	CS	E	+	+								1.13	N	N	A
85	112	33	0.8	N	14	3.5	N		HI	+									1.21	N	-	A
86	84	34	0.8	N	12	3.1	N	DTS	BHI	+									1.12	HI	-	D
87	111	28	0.7	N	13	2.3	-		HI		+								1.27	HI	-	A
88	234	110	3.2	N	9	2.4	-		NHI		+								1.27	N	-	A
89	96	42	1.1	N	12	2.6	-		MS/ME	+	+		+	+					1.15	HI	-	A
90	99	32	1	N	13	2.8	p/c/x	EDS	HI		+								1.18	N	-	A
91	103	28	0.8	N	7.8	2.4	C		HI/E	+	+				+				1.15	HI	-	A
92	102	26	0.7	N	8	2.8	-		HI	+									1.13	N	-	A
93	121	82	1.8	N	15	3.6	-		ME/E		+								1.21	HI	-	D

94	109	30	0.8	N	13	3.1	-		HI	+									1.12	N	-	A
95	106	28	0.7	N	14	2.6	C		MS/E		+								1.27	HI	-	D
96	98	26	0.7	N	10	3.4	N		HI	+									1.27	N	N	A
97	120	32	0.9	N	7.2	2.9	-		ME/NS	+									1.37	N	N	D
98	131	36	1	N	7.6	3.4	-	N	N		+								0.9	N	-	A
99	85	40	1.2	N	8.4	2.4	-		HI	+									1.22	N	ACL	A
100	76	44	1.1	N	9.6	3.1	N		HI										1.11	N	N	A